VOLUME 58 NUMBER 5

# ARCHIVES OF SURGERY

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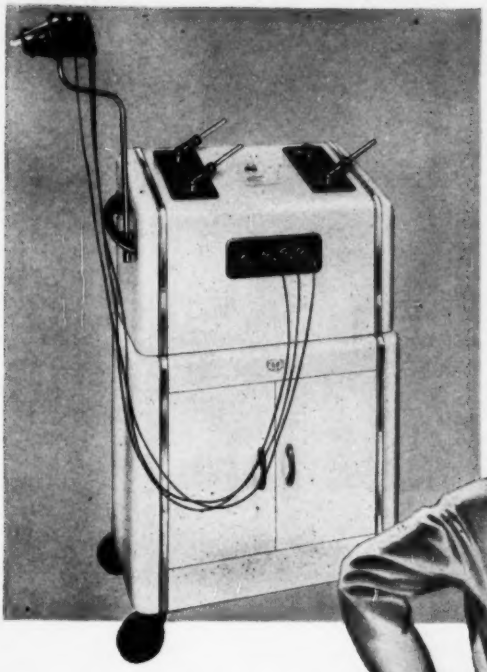

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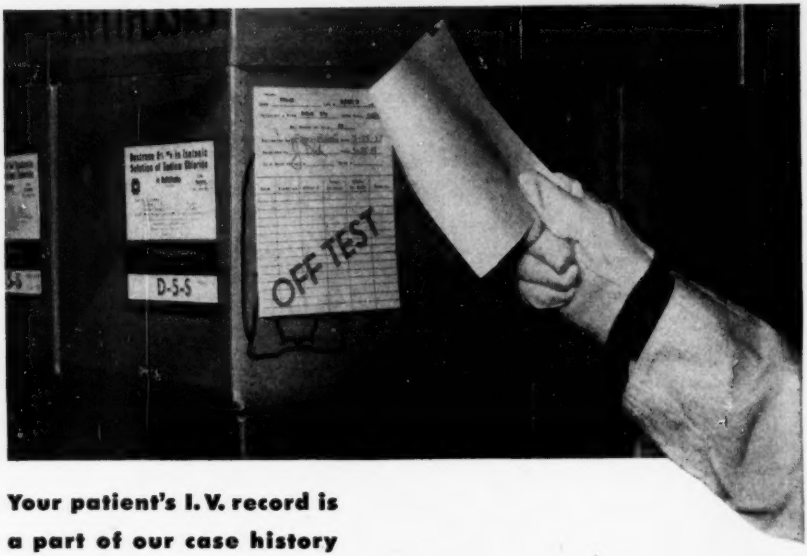


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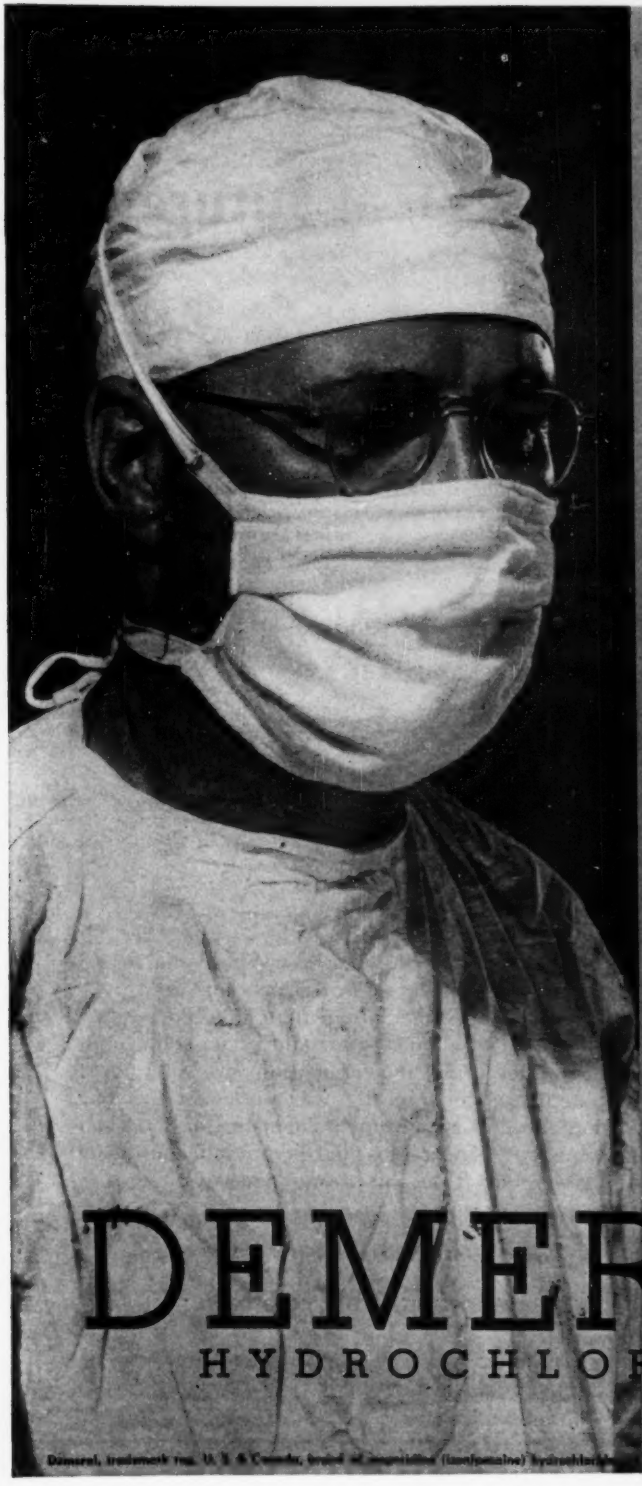

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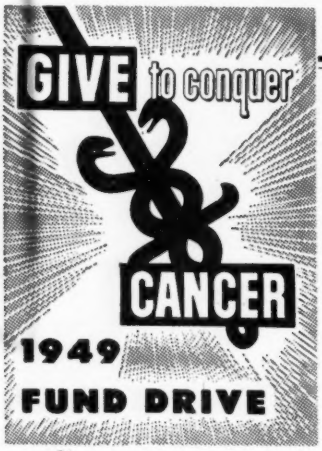


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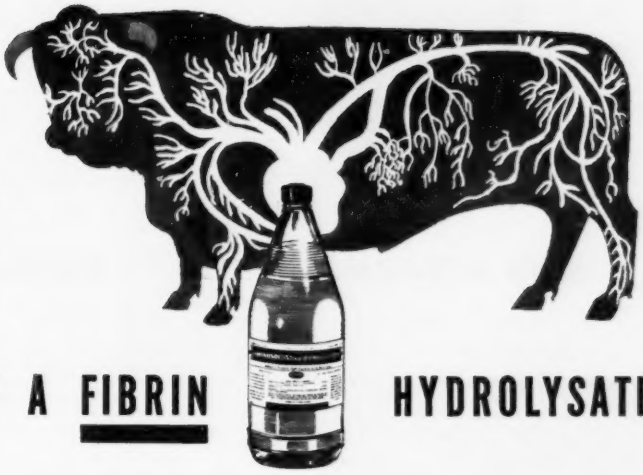

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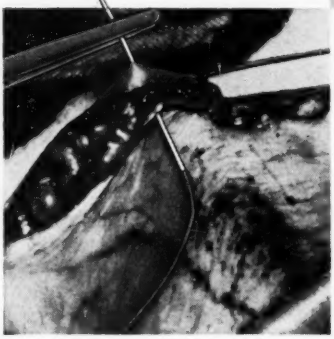

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ARCHIVES OF SURGERY

VOLUME 58 MAY 1949 NUMBER 5

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# MORE HUMAN ATTITUDES IN SURGICAL PRACTICE

**Presidential Address**

HARRY B. ZIMMERMANN, M.D.
ST. PAUL

PSYCHOLOGISTS tell us that there is no such thing as a mentally absolutely normal human being, that some of us tend toward aberrations paranoid in type and that some of us are a trifle schizoid. I must admit, after thinking over what I am going to present today, that I belong to the latter group. So I hope you will bear with me and try to realize that the things I say are abstract generalizations and that you will not take the attitude of the wife who once asked her husband why women were always excluded from males in after-dinner conversation. He told her that the discussions were usually more or less controversial, but definitely dispassionate, and that women tend to take abstract criticism personally and immediately become defensive. Her response to this was, "But, John, I'm not like that, am I?"

The Preamble of the Constitution of the Western Surgical Association reads, in Article 2: "The object of the Association shall be the cultivation, promotion and diffusion of the knowledge of the art and science of surgery; to sponsor and maintain the highest standards of practice."

There is no doubt that we have accomplished great things and filled the obligations of the first portion of this Preamble beyond the fondest dreams of the founders of the organization. In going over the *Transactions of the Western Surgical Association,* one will be struck by the fact that a great deal of the development of the art and science of surgery has been accomplished by our members and that at each meeting surgical diseases have been studied from the viewpoints of pathogenesis and pathology and expedient technical procedures for their alleviation and cure have been elaborated and discussed.

But have we done so well, or spent enough of our effort, on the last phrase of the preamble—"to sponsor and maintain the highest standards of practice"? No one can doubt that the Western Surgical Association has maintained the highest of surgical practice, but that we have made any active or vigorous effort to sponsor or assume any leadership (except by our own examples) in developing the highest

Read at the Fifty-Sixth Annual Meeting of the Western Surgical Association, St. Louis, Dec. 3, 1948.

type of surgical practice for the good of the community or gone out of our way, appreciably, to make available to the communities in which we live the highest type of surgical practice is open to question.

This discussion was motivated by a series of articles, both in lay magazines and in medical journals, criticizing surgeons for doing unnecessary and ill advised operations. An article in the *Woman's Home Companion* in July 1948 began with the statement: "Medical men have long known the shocking fact that many of the nine million surgical operations performed annually in America are unnecessary. Among doctors it is an open secret that in many an operating room the cloak of surgery covers mayhem and even manslaughter." In this same article a well known gynecologist is quoted from an article in the *American Journal of Obstetrics and Gynecology* under the title "Hysterectomy: Therapeutic Necessity or Surgical Racket?" He analyzed 246 operations in which the uterus was removed and in which 30 per cent of the removed uteri were absolutely normal. Also in this article was a quotation on the same subject from an editorial in *The Journal of the American Medical Association.*

In a recent number of the *Reader's Digest* a story was epitomized from *The Modern Hospital* entitled, "The Truth About Fee Splitting." Again, in the October issue of the *Woman's Home Companion* there was a story, read by thousands of our citizens, lamenting the predicament of the thoroughly qualified young surgeon who has had five years resident training, is entirely qualified to do any kind of surgery and whose talents are denied society because he cannot obtain hospital facilities absolutely essential to do his work, and this because of the selfishness of less competent older surgeons.

Society recognizes that scientifically and technically we have accomplished much and considers our services indispensable. It is genuinely appreciative of our accomplishments, as evidenced by the fact that there has never been a time in history when the medical and surgical professions have been more abundantly rewarded, not only by generous individual remuneration but also by vast contributions for medical education, graduate study, almost extravagant hospital facilities and huge funds for research. In other words, the criticism in lay periodicals accuses us solely of not living up to our traditional reputation for altruism and devotion to the service of mankind.

Let us, then, take these various accusations specifically. Are we doing unnecessary and ill advised operations? In editorials on this subject in our own journals we have already pleaded guilty. Such a plea precludes any defense, but we should make an effort to correct this situation.

This is not so easy, because there are many conditions which have brought it about. Broadly speaking, it seems to me that these may be put into three classes: first, plain dishonesty, avarice and cupidity among a small proportion of the members of our profession; second, ignorance of therapeutic procedures other than surgical ones and, third, too great a zeal for technical surgical achievement.

With regard to the first, we will always have members of the profession who will find it difficult to live up to its ideals, but at least we can improve the situation by maintaining our ideals and accentuating their importance. They are the oldest traditions of the medical profession, and from time immemorial it was considered important to indoctrinate the candidate for medical practice with a certain moral code and a proper appreciation of his obligation to society. The apparent lack of ethical sense can only be obviated by (1) selecting more carefully candidates for medical school, (2) seeing to it that they have an opportunity for a broader cultural and humane education and (3) indoctrinating them, all during their training, with proper ethical values.

Better selection of the types of persons admitted to medical schools is difficult because medical faculties do not select their students. Candidates for medical schools make up their minds they want to become physicians, or their parents make up their minds for them. Very often, and properly, medicine is selected as a career for a young man in order that he may improve his social and economic status. This is a laudable motive; equal opportunity is the greatest blessing of a real democracy, and opportunity to achieve social betterment should be denied no one. But the person who goes into medicine solely to improve his social and economic status will more than likely accomplish that sole objective.

There is probably no one less competent to appraise the aptitudes of a youngster for the profession of medicine than are his parents. All of you have had a fond mother tell you that little Willie is to be a surgeon and that she knows he will be a success, because he has always loved to pull things apart, especially bugs and grasshoppers, and that now he is in high school he is just fascinated with the business of cutting up cats. But his brother Jim just never could be a doctor—he is much too sensitive. He never could bear to hurt things. So he is going to be a minister, a servant of the Lord.

I feel like asking such a mother if little Willie had ever expressed a desire to become a pirate or a bandit and, if he had, telling her she would make a great mistake in trying to thwart this very natural ambition! As for Jimmy, would she please suggest to him that many

of our greatest surgeons selected their professions purely as a means of serving the Lord?

In order that there may be some opportunity for the medical faculty to take part in the selection of their students, I think it is important that we go back to an academic requirement of from three to four years and discontinue the "pre-medical" school. Up to a few years ago it was required that a medical student have this period of academic or cultural training; now this has, to all intents and purposes, been entirely eliminated. Young men are admitted from high school to what is termed "pre-med." In this two year premedical course they are intensively taught the basic sciences related to medicine; any cultural training they get during this time is purely incidental. The assumption today is that when a young man enters premedical school he is already a candidate for his medical degree and, unless he is expelled or fails, he will receive it. Should he fail before the end of his second year, so far as he is concerned the two years will have been wasted.

Substituted for this premedical education should be a general education of from three to four years in the liberal arts college. Here the student should begin a system of ethical indoctrination. It is at this stage that the various psychologic aptitude tests might be applied, as well as tests to discover important intellectual deficiencies. For instance, there would be many candidates who would be found to be functional illiterates, having not yet learned to read with speed and comprehension. Others, because of the great variation in home cultural backgrounds, might be handicapped, and they should be given special opportunities. Colleges preparing students for medical school should have available an adequate staff of carefully selected, mature advisers—not young men, candidates for doctors' degrees in limited fields, or recent recipients of such degrees, but mature, well educated persons qualified to appraise the shortcomings and talents of students and to advise them properly as to courses that would best develop them. With the present overcrowding of our educational institutions, it might be difficult to find enough such personnel, but I think it would be feasible to recruit advisers from outside the university as volunteers and train them for their jobs. Their function would be to select courses to overcome the aforementioned deficiencies.

There was a time when medicine, law and theology were considered learned professions. These professions were taught in universities and were honored with doctors' degrees. The secondary school preparation for them was in the classical humanistic tradition, in this country by the latin school, in England the public school, in France the lycée and in central Europe the *Gymnasium*. When technical skills became essen-

tial to society, the polytechnic schools were established. These schools differed from universities in that graduates usually did not receive doctors' degrees. Their function was to train experts in chemistry, engineering, agriculture, military science and various other technical skills. The *Realschule* was the preparatory school for the polytechnic institute; it was a sort of revolt against classical humanism. It taught the essential humanities, but mathematics and the natural sciences were emphasized. There is no doubt that classical humanism came to be a type of intellectual snobbery that we have properly outgrown, but I feel that today we are making a fetish of educational realism. When a degree of doctor of medicine is merely a license to practice a service trade along with druggists, optometrists, masseurs and so forth, we should not be shocked if its recipients have big business or craft guild objectives.

One thing which is extremely important is that teachers and advisers, as well as medical students themselves, be oriented as to the importance of science. It is necessary that they get over the notion that physicians, and especially surgeons, are scientists. It is vital that all medical men have a thoroughly competent scientific background; science is basic to the understanding of a physician's profession. A little boy once defined salt as "the stuff that makes potatoes no good when you don't put it on." Philosophy and the humanities are the salt for science. A great scientist, and one of the foremost biochemists of today, President Conant of Harvard, points out how rarely in our daily lives we are influenced by the results of modern science but how often we reflect in our acts the philosophy and poetry we have imbibed over many years. "A dictator," he says, "wishing to mold the thoughts and actions of a literate people could afford to leave the scientists and scholars alone, but he must win over to his side, or destroy, the philosophers, the writers, and the artists."

I am using the term science in its generally accepted connotation: knowledge that lends itself to a strictly quantitative formulation. But there are two adjectives commonly used to designate intellectual values, always in a negative or exclusive sense: first, "scientific," having a kind of myopic acquaintance with laboratory method, and, second, "practical," a sort of empiric approach that gets by without much of any knowledge. Neither of these exclusive qualities is in the least critical for the selection of candidates for the practice of medicine.

Scientists must have great information, and they must know how to apply it—which is knowledge. This information must be accurate: it need not necessarily be adequate in the sense that it should be for the surgeon. The surgeon must be wise, and I use the word wise advisedly. Webster's definition is "discerning, and judging soundly

concerning what is true or false, proper or improper; choosing the best ends and the best means for accomplishing them."

To make a physician's knowledge adequate, he must have comprehension of those things inspiring human action; he must have had, some time in his life, the benefit of contact with, and observation on, the motives and stimuli of man's behavior. He needs to acquaint himself with all aspects of human nature. The surgeon who has been trained to recognize that his patients are human beings and have lives beyond the operating table will not conclude his efforts without reflecting on the effects of these efforts on the lives of his patients. His knowledge is inadequate, and he is not wise, if he ignores the emotional differences of men. Scientists are not required to emphasize this point, but surgeons are. Persons who are preparing for medicine or surgery and who will have to deal with the destinies of human beings must have humane knowledge. Their education must be liberal, humanistic and man centered.

The academic training for medicine should not be intended to make successful contestants for radio quiz programs by filling the student with all sorts of information. Our students need information, they need knowledge more and they need wisdom most of all. They should have a sufficient knowledge of such subjects as history of science, history of ideas and those manifestations of human emotions such as religion, literature and the arts, sufficient of philosophic method to give them assistance in knowing the nature of proof, the criteria of valid judgment, the relation of knowledge and action, the difference between infantile and sophisticated thinking and other information tangential to the making of adequate judgments.

It is all important for a physician to be able to pass sound judgment, to evaluate adequately and to know with reasonable certainty how to make the ever finer distinctions and to do all this humanely. Yes, we need information, but we need knowledge and wisdom most of all. The selection of young persons competent to study medicine is essential for the future of our profession. Three to four years of college training is extremely important—important for the student in that it gives him an opportunity to acquire values necessary for the study and, later, the practice of medicine and important for the medical faculty in that it gives them an opportunity to make aptitude and personality inventories necessary for the selection of students who should be encouraged to take up medicine as a profession.

As an illustration of what I mean by ethical indoctrination, this story was told me by a professional educator, one time President of St. John's College in Annapolis, Md. Being at Annapolis, he was familiar with the training methods at the naval academy. He had

observed the rigid, severe discipline, the incisive and monosyllabic answers given to questions by the neophyte. All this, he understood, was to train the young cadet in unquestioned obedience. On a visit to England he had an opportunity to look over a British naval academy. There he found little of this formal discipline. The students did not march in military formation to and from classes, and they were relaxed and at ease but behaved like gentlemen at all times. He learned that the British midshipman began his training much earlier than the American one, and during his entire academic life, along with his technical training, he was actually brought up on the heroic poetry and stories of England and especially of the navy. From this type of literature he became so imbued with the heroic traditions of the British navy that his loyalty and obedience would never be questioned.

As to the medical school itself, there has never been a time when medicine was as competently taught as it is today in our accepted medical schools, but there are some phases of the teaching that could be improved on. There is a tendency on the part of the medical faculty to treat undergraduate medical education as a preparatory training that would be more or less valueless if it were not supplemented by two to five years of graduate work. In the four years at medical school the student should be taught all branches of medicine, with emphasis on the various clinical studies in proportion to their importance for a good general practitioner. The student should never be allowed to lose sight of the fact that he is being trained to be a physician and that his first duty is to his patient.

The importance of the various clinical studies is a matter for the faculty to decide. Because the student has decided early in his career that he intends to become a specialist, he should not be allowed to neglect certain fields because *he* feels that they are unimportant to him. If we are to expect a proper and unprejudiced attitude on the part of the student, that attitude must be most scrupulously manifested by the clinical teacher. He should accept the fact that medicine is one and that the basic principles underlying its teaching are the same for all clinical fields. It must be definitely understood that specialization should be inclusive and not exclusive; if one acquires special knowledge or skill in some field of medicine it should be in addition to, not at the expense of, basic knowledge of medicine. It is not only expedient, but proper, that the various clinical subjects and therapeutic skills be taught by specialists, but it is poor pedagogics to overemphasize one field to the disparagement of others. This attitude on the part of a teacher will confuse the student and spoil his concept of oneness in medicine.

As for the intern year, this could be one of the most important periods in the education of a physician. The intern is in the most impressionable stage of his medical life; he is an abject hero worshiper. Not only do we take no advantage of this character to develop the best that is in the intern, but we neglect and discourage any spark of idealism he may have previously acquired.

Assume that a young graduate in medicine, possessing air castles built of the best ideals on a foundation of altruistic ambitions, has received an appointment as an intern in a teaching hospital. His hope is that now he will come to know the men he has listened to from the benches in the amphitheater. He asks for nothing more than to be a disciple of these great men for a year, to learn their clinical methods, to study patients and to be able to submit the results of his studies to the master for his approval, and should he occasionally receive a word of commendation, his ego will be built up exceedingly. But this is not what happens. He is almost never allowed to speak either to the professor or to the patient, he receives orders from a third assistant resident, written on slips of paper or over the house telephone, to pass a catheter or a stomach tube, insert a needle in a vein or deliver some material to the laboratory for analysis. He is on what is known as a "straight service." If this is a surgical service, he may hold a retractor during an operation. He may even be allowed to do a small operation under the direction of the resident. What a perverted idea such a young man gets of the practice of surgery. He receives no intellectual stimulus. His entire internship is spent in a series of mechanical doings. He passes tubes, he inserts catheters, he works in the operating room holding retractors; later on he is rewarded by being allowed to do some small operation. This entire experience is not only uninspiring but disillusioning and heartbreaking. Should this young man be one of the eighty or ninety in a hundred who will have to go out into the world to practice, with no further clinical knowledge of the practice of medicine and a most perverted notion of surgery, is it any wonder that he may lose some of his ideals? Should he have received his internship in a private hospital, he might at least have had a varied and more adequate clinical experience, but if we are to take as factual the statement in the *Woman's Home Companion* that "thirty per cent of all operations are done for the benefit of the surgeon only," then this training certainly will not conduce to the best in medical practice by him.

It will be a long time before we can develop any standard for graduate surgical training. It is a question whether too much standardization is to be desired. If one realizes that graduate training in special fields is new in this country, it must be admitted that we are doing remarkably well.

I said earlier in this paper that I felt that ignorance of therapeutic methods other than surgical ones was responsible for a great deal of ill advised operations. None of us enjoy being regarded as pure craftsmen who perform mechanical operations according to the prescription of physicians; we resent having surgical therapy prescribed as is physical therapy or the compounding of medicines; we contend that we are physicians, able to make or verify diagnoses and to evaluate the indications for surgical therapy. This attitude on the part of the surgeon would be right and reasonable if his premise were true. There are practically no diseases today wherein surgical treatment is never indicated, and if the surgeon assumes competence for making his own diagnoses, he must be trained to have this competence. If he expects recognition for being able to evaluate the advantages of surgical treatment over other methods, he should know, unprejudicially, the possibilities and value, if not necessarily the technics, of all other treatments. This should be the attitude in all fields of special therapy, and with this concept we should indoctrinate the graduate student. I sometimes feel that we give students the impression that nonsurgical treatment, if not purely palliative, at least is not permanent—that there is a finality only in ablative surgery. If by that we mean that, once done, ablative surgery cannot be undone, this is true.

The teaching attitude in some of the larger cancer centers in Europe, and in one or two in this country, appeals to me. The resident serves in all the therapeutic departments of the institution and becomes sufficiently expert in all forms of therapy, so that, without prejudice, he can use with wisdom any form of treatment to the best advantage of the patient. I feel that we should have frequent conferences of all the special groups, physicians and surgeons, to study diseases and their treatment in which we all might have an interest.

There is no reason today why we must perpetuate the medieval, academic feud between physician and surgeon. In the graduate training for both medicine and surgery, cooperation between the physician and the surgeon and the psychiatrist is not enough; there should be more actual collaboration. In any hospital with which I am associated we do not even have any consultations with other departments. The modus operandi is this: The physician who happens to be in charge decides that the patient is suffering from a disease or a complication not in his field, so he orders his resident to have a physician or some specialist see the patient. The so-called consultant sees him, writes an opinion or accepts him for treatment, and that is that. There is no conference and no consultation. This procedure is expedient and often the only one that is possible, but it is poor training for a resident. It should occasionally be possible to have the consultants, in a conference, discuss what procedures might be best for patients.

The following is a quotation from a paper given by Dr. Barney Brooks before the St. Louis Medical Society in May 1947:

> Six years ago, I committed what even some of my most loyal associates considered rank heresy. I brought a psychiatrist and a psychologist into the surgical wards. This was done not only because of an idea I got that the surgical wards were perhaps the place best suited for clinical research on some of the problems of psychiatry, but principally for the purpose of stimulating surgeons-in-training to extend their interests into the psychic as well as the somatic aspects of disease. I have been amazed at the change of viewpoint of even the conscientious objectors who felt quite sure patients would be insulted by even the thought of having to see a psychiatrist to the point of leaving the hospital immediately. There is no doubt in my mind that this venture has influenced greatly my House Staff to think of a patient as a human being rather than a hunk of nonprotein nitrogen and blood sugar without enough hydrogen ion concentration to absorb an autolysing appendix. It has also emphasized the importance of the surgeon's knowing at least a little about a great many things to be able to commune with anyone. A brief mutual understanding may establish a personal relationship between that patient and the surgeon which is of great value for diagnosis and even greater influence for obtaining the most favorable result.

Too great a zeal for technical achievement is often a vice in an otherwise well equipped surgeon. The well trained airplane pilot must be equipped to do all manner of stunts and aerial acrobatics. He must be taught to stall his plane, to go into a spin, recover and level off. All of these are hazardous, but any of the maneuvers may some day save lives. On the other hand, the pilot who drives his plane day in and day out in such a way that these performances are unnecessary is by all odds the best pilot. The zealous exhibitionist who rates his ability by the accomplishment of the spectacular is dangerous. The training of surgeons is analogous. We should teach the young surgeon all the surgical technics that he may some day find necessary to use, but we must also teach him, and by personal example, that too great a zeal for spectacular technical achievement is dangerous and could be interpreted as a means for the acquisition of unwarranted publicity.

Underlying all definitions of education is the concept that it denotes an attempt on the part of adult members of society to shape, in accordance with their own ideals, the development of the coming generation. To this extent we are all educators.

The membership of the Western Surgical Association can be roughly divided into two groups—those who have chosen to follow an academic career and those who in the main are engaged in private practice. The function of the teaching surgeon is to teach. The surgical education of medical students, interns and graduate students specializing in surgery is his first responsibility. He must also be able to contribute to the progress of surgery by his promotion and supervision of intelligent surgical investigation. His judgment must

be critical and unprejudiced in evaluating research. Should he acquire some unusual knowledge or skill in surgery, he should be encouraged to use it for the benefit of the sick and receive compensation from those who can afford to pay him, but he should not use his time, which belongs to his students and co-workers, to perform such surgical procedures as could be done as well or better by other surgeons in the community in which he lives. The criteria of his achievement are competent, honest surgeons whom he has trained and a progressive, productive department of surgery developed under his guidance.

The private practicing physician, especially if he intends to remain in private practice, must also concern himself with his social function. He must do more than give lip service to the Hippocratic Oath and the obligation of the American College of Surgeons. He must realize that in a broader sense he too is an educator in that as an adult member of society he is responsible for the oncoming generation of surgeons and their ideals. Any prestige he may acquire from a grateful following in his community he should use to foster better surgery, better hospital facilities and a better surgical personnel to take over when he must retire.

Right now he is most concerned with what is to become of him and the private practice of surgery. He is fearful to the point of a severe anxiety neurosis. I personally am anxious about, and look with suspicion on, any attempt by society or the government to interfere with the practice of surgery or my way of life, but I must admit that we ourselves are somewhat responsible for the rebellion.

At a meeting of the Rural Health Cooperatives, held in Minnesota, an address was made by an Assistant Secretary of the Interior. He took the position that the recent election was a mandate by the people for medical social reforms, and the sense of the meeting was that the social side of medicine is properly the concern of educators and social philosophers and not of physicians. A prominent Catholic educator, who obviously could not be accused of leaning, even slightly, toward communism, made the categoric statement: "Medical scientists should not invade the social field any more than educators and social philosophers should invade the scientific field."

If his premise is true, that physicians are pure scientists and technicians, completely ignorant of humane social philosophy, his thesis is incontrovertible. But I do not believe that his premise is true. If society has such an opinion, it is our business to change it. I think that this can best be done by interesting ourselves, actively, in the humane and social aspects of our profession and, throughout the entire period of their training, making every effort to instil and develop in the oncoming generation of physicians a recognition of the importance of these attitudes.

# REVASCULARIZATION FOLLOWING EXPERIMENTAL MESENTERIC VASCULAR OCCLUSION

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DETROIT

THE SMALL intestine has a remarkable ability to withstand interruption of its blood supply. The distribution and anastomoses of its vessels appear to be responsible for its great revascularizing potential, and it has been generally assumed that the peculiar arcuate system of the intestinal tract is the chief factor in overcoming vascular deprivations. Considerable experimental effort has been devoted to determining the degree of vascular interruption which is compatible with life, yet the exact mode by which compensation takes place has not been clearly established. The literature pertinent to these studies may be discussed under two categories—(1) anatomic studies and (2) experimental mesenteric vascular interruption and revascularization of the intestine.

## ANATOMIC STUDIES

Morphologic reports on the larger vessels of the small intestine are in accord, but many discrepancies appear in descriptions of the vasa recta and the vessels within the intestinal wall. Much of the confusion doubtless arises from the common failure to recognize the fact that striking species differences exist. Injection studies reported from these laboratories in 1943 [1] clearly show that the pattern followed by the intestinal vessels of the dog, for example, differs greatly from that of man. While most species studied fall into one or the other of these two patterns, the variations are striking and sufficiently great to cause confusion if one makes the common error of assuming that a single intestinal vascular pattern is common to all species.

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1. Noer, R. J.: The Blood Vessels of the Jejunum and Ileum: A Comparative Study of Man and Certain Laboratory Animals, Am. J. Anat. **73**:293, 1943.

Dwight in 1898 and 1903 [2] presented a brief but clear description of the mesenteric vascular arch system in man and reported that the vasa recta do not intercommunicate until the intestinal wall has been reached. Monks in 1905, 1909 and 1929 [3] published a series of classic studies of the anatomy of the human small intestine. His papers are chiefly concerned with the mesenteric vessels and their utilization in small intestinal localization; the increasing complexity of the arcuate vessels in the more distal portions of the human small intestine is well portrayed. It is of particular interest that Monks' illustrations have been reproduced from time to time, redrawn in such a way as to show a nonexistent human mesenteric border artery which is neither illustrated nor described in his original papers.

Eisberg in 1924 [4] reported a thorough study of the mesenteric vasculature of both man and dog. Though his illustrations clearly show the differences between these species, he fails to point out that there are communications between the dog's vasa recta but none in man. This paper is particularly noteworthy for its description of the vessels within the intestinal wall and clearly shows the abundant communications which exist between the mural trunks, though it fails to point out the differences between species with respect to these anastomoses.

Mall in 1887 [5] presented a detailed description of the blood and lymphatic vessels of the small intestine of the dog. His account clearly delineates the pattern of the mesenteric arcades, the abundantly intercommunicating vasa recta and the anastomoses between the mural trunks. His descriptions of the finer ramifications of the arteries, veins and lymphatic vessels within the intestinal wall are complete in finest detail, and the paper is a masterpiece unequaled elsewhere in the literature on this subject.

Latarjet and Forgeot in 1910 [6] described the circulation of the jejunum and ileum in man, dog, cat, horse, pig and rabbit. This is the most complete early comparative study, and the observations are

2. Dwight, T.: The Branches of the Superior Mesenteric Artery to the Jejunum and Ileum, Proc. A. Am. Anat. **10**:79, 1898; The Branches of the Superior Mesenteric Artery to the Jejunum and Ileum, Anat. Anz. **23**:184, 1903.

3. Monks, G. H.: Studies in the Surgical Anatomy of the Small Intestine and Its Mesentery, Ann. Surg. **42**:543, 1905; Intestinal Localization, J. A. M. A. **52**:1079 (April 3) 1909; Intestinal Localization, Surg., Gynec. & Obst. **49**:213, 1929.

4. Eisberg, H. B.: Intestinal Arteries, Anat. Rec. **28**:227, 1924.

5. Mall, F. P.: Die Blut-und Lymphwege im Dünndarm des Hundes, Abhandl. d. math.-phys. Cl. d. k. sächs. Gesellsch. d. Wissensch., 1887, vol. 14.

6. Latarjet, A., and Forgeot, E.: Circulation artérielle de l'intestin grêle (duodénum excepté,) chez l'homme et les animaux domestiques, J. de l'anat. et physiol. **46**:483, 1910.

carefully presented. These authors stated that they consider the embryologic background an important factor in the adult situation.

Bradley in 1927 [7] gave a brief but accurate account of the mesenteric system in the dog. DeBlasi in 1937 [8] compared the distribution of the superior mesenteric vessels of dog and man. His report included a description of multiple mesenteric occlusion experiments which led him to the conclusion that the human intestine is more efficient in revascularization than that of the dog.

Dieulafé in 1936 [9] reported on injections of the mesenteric vessels of the dog and rabbit with radiopaque mediums. It was his conclusion that the dog possessed a much greater degree of vascularity than the rabbit. Morton in 1929 [10] discussed the relative vascularity of the duodenum and ileum of the dog indicating that the more proximal portions of the intestine possess not only larger mural vessels but also more extensive anastomoses between these radicles. (Data reported from this laboratory in 1943 [1] confirm this finding.)

Comparative studies of the small intestinal circulation in man and certain laboratory animals were reported from this laboratory in 1943.[1] This report clearly demonstrates the variations among species, with respect to both the larger vessels and the intramural circulation. So far as we know it presents the only complete description of the types of anastomoses within the wall of the intestine in various species. Since this paper was published, additional studies have been made of the rat and the hamster, which indicate that the mesenteric pattern of the rat is similar to that of man, while the hamster, in at least one respect, differs from all the species so far studied.

Special consideration should be given to two frequently quoted papers concerning the blood vessels of the small intestine. The first of these is that by Cokkinis [11] published in 1930. This author carried out injection studies on human autopsy specimens, using india ink, and his illustrations clearly show the results obtained. It is of particular interest that he stated: "The collateral circulation stops with the terminal row of arcades. Beyond this there is absolutely no anastomosis, either between the vasa recta in the mesentery or between the

7. Bradley, O. C.: Topographical Anatomy of the Dog, London, Oliver & Boyd, 1927, pp. 27-28.

8. DeBlasi, A.: Occlusione dei vasi mesenterici ed infarto intestinale, Gazz. internaz. med.-chir. **47**:95-107, 137-151 and 247-255, 1937.

9. Dieulafé, R.: Contribution à l'étude de l'irrigation de l'intestin grêle-Conséquences de la ligature expérimentale des vaisseaux mésentériques, Arch. d'anat., d'histol. et d'embryol. **21**:97, 1936.

10. Morton, J. J.: The Differences Between High and Low Intestinal Obstruction in the Dog, Arch. Surg. **18**:1119 (April) 1929.

11. Cokkinis, A. J.: Observations on the Mesenteric Circulation, J. Anat. **64**:200, 1930.

ramifying vessels on the gut wall. One feels that the last observation contradicts the view of many authors but its accuracy is guaranteed." There can be no doubt, from his illustrations, that his injections failed of complete filling of the finer vessels, but this fact hardly justifies his emphatically stated conclusion that there are no anastomoses at any point distal to the arcuate vessels. Most observers are in complete agreement as to the existence of such anastomoses, and the papers of Dwight,[2] Mall[5] and Eisberg[4] clearly demonstrate them. The report previously referred to[1] contains photographs of injected specimens, including flat mounts of the antemesenteric area which can leave no doubt of the presence of abundant intercommunications between the mural vessels (fig. 3). Proof of this anastomotic mechanism is further adduced in the body of this paper.

A second widely quoted paper is that of Dragstedt, Lang and Millet, published in 1929.[12] These authors expressed the opinion (since then widely accepted as fact) that the vessels of the intestinal wall pierce the muscularis at levels progressively farther from the mesenteric attachment in the more distal portions of the intestine and postulated that this might be an explanation for the greater morbidity and mortality associated with high intestinal obstruction. The paper from this laboratory previously referred to[1] reported on serial sections of injected specimens prepared with special stains in man, dog, chimpanzee and opossum, with a total count of the vessels piercing the muscularis at various levels in both jejunum and ileum. There was no significant variation between the levels of the intestine with respect to the point at which the vessels pierced the muscularis. On the basis of these studies the aforementioned hypothesis appears to be no longer tenable.

## EXPERIMENTAL MESENTERIC VASCULAR INTERRUPTION AND REVASCULARIZATION OF THE INTESTINE

The literature contains many references to these subjects, but in only a few are the data sufficiently controlled to justify detailed consideration in this report. One of the earliest and most carefully prepared studies was that of Welch and Mall in 1897, not reported in detail until 1920.[13] It is of interest that this paper is often misquoted on the basis of incomplete data published in Allbutt's "System of Medicine," 1902 and 1909.[14] From misinterpretation of this work has grown the

12. Dragstedt, C. A.; Lang, V. F., and Millet, R. F.: The Relative Effects of Distention on Different Portions of the Intestine, Arch. Surg. **18**:2257 (June) 1929.

13. Welch, W. H., and Mall, F. P.: Experimental Study of Hemorrhagic Infarction of the Small Intestine in the Dog, in Welch, W. H.: Papers and Addresses, Baltimore, Johns Hopkins Press, 1920, vol. 1, pp. 77-109.

14. Allbutt, T. C.: A System of Medicine, New York, Macmillan & Co., 1909, vol. 6.

erroneous concept that 5 cm. is the maximum length of small intestine in the dog which will withstand mesenteric devascularization. These authors actually showed a high percentage of survival in animals subjected to devascularization of 13 cm. loops. It is of particular significance that they pointed out that some of their data clearly indicate that the reestablishment of circulation in some of these animals could have taken place only through the intramural vessels.

Additional experiments on mesenteric occlusion have been reported by many authors, among them Demel,[15] Eisberg,[16] Murphy and Vincent,[17] Dieulafé,[18] Niederstein[19] and others. These investigators have shown the effects of various types of mesenteric interruption, and although many of their conclusions differ, they are apparently in agreement on the following ideas: first, that in the dog the maximum length of small intestine that will survive mesenteric devascularization is 5 cm.; and, second, that venous occlusion produces a congestive necrosis more hazardous to the animal than arterial deprivation. There is, however, no uniformity of opinion as to the validity of comparing these results with similar situations in man.

The literature on experimental mesenteric occlusion was briefly reviewed and additional data reported from this laboratory in 1948.[20] It was shown that interruption of any or all of the mesenteric blood vessels to a 15 cm. segment of the dog's small intestine was followed by death in less than half of the animals so treated. It was further shown that complete vascular deprivation of such a segment could not be uniformly accomplished by interruption of intestinal and arcuate vessels alone. These results supported the belief that revascularization had presumably been accomplished through anastomotic connections within the intestinal wall and/or by way of the marginal plexus peculiar to the dog. Injection studies carried out subsequent to this report have further showed that 15 cm. is likewise the maximum segment

15. Demel, R.: Zur Frage der Ernährung des Darmes bie Gefässunterbindungen im Mesenterium, Arch. f. klin. Chir. **146**:701, 1927.

16. Eisberg, H. B.: Viability of the Intestine, Proc. Soc. Exper. Biol & Med. **21**:153, 1923; Viability of the Intestine in Obstruction, Ann. Surg. **81**:926, 1925.

17. Murphy, F. T., and Vincent, B.: Experimental Study on the Cause of Death in Acute Intestinal Obstruction, Boston M. & S. J. **165**:684, 1911.

18. Dieulafé, R.: Recherches expérimentales sur certaines lésions segmentaires de l'intestin grêle: infarctus hémorragique, gangrène, iléite, Progrès méd. **67**:449, 1939.

19. Niederstein: Die Zirkulationstörungen im Mesenterialgebiet, Deutsche Ztschr. f. Chir. **85**:710, 1906.

20. Derr, J. W., and Noer, R. J.: Experimental Mesenteric Vascular Occlusion, read before the Forum on Fundamental Surgical Problems, American College of Surgeons, 1948; Surg., Gynec. & Obst., to be published.

of comparably devascularized dog's intestine which can uniformly be filled with injection mediums. Longer segments, which caused death of the animals, similarly failed to fill when injected. In other words, there appears to be a fairly accurate correlation between the length of segments which can be filled by injection and those which are compatible with life after mesenteric vascular interruptions. This method of study may, therefore, be useful with reference to man, in whom experiments are impossible during life.

One clinical report appears most significant with respect to the revascularizing ability of the human intestine. Bost in 1929 [21] presented 3 cases in which patients suffered complete detachment of segments of the small intestinal mesentery varying in length from 4 to 8 inches (10 to 20 cm.). The mesentery was resutured and omentum wrapped about it, after which the patients made a complete recovery. He also reported three experiments of this type in dogs, 2 of which survived after resection of 6 and 8 inches (15 and 20 cm.) of mesentery respectively. In the same year Rothschild [22] presented a report on a series of injections in the dog's small intestine which indicated a considerably greater revascularizing power than had formerly been believed. He also expressed the opinion that man possesses a greater factor of safety than does the dog.

## EXPERIMENTAL DATA

Repeated arterial injections have been carried out with two technics: (1) liquid latex and (2) india ink. The latter provides a more easy and complete filling of the finer radicles at lower pressures and has accordingly been utilized for most of the studies. Bovine plasma has been utilized to wash the blood from the vessels prior to injection and has routinely been used as the diluent for the ink. Davies and Edwards [23] have called attention to some of the advantages of plasma for injection mediums, and we have found that its use eliminates the edema so often produced by preliminary irrigations with isotonic sodium chloride solutions. Latex specimens were "set" in acetic acid, then fixed in 10 per cent solution of formaldehyde, while the india ink preparations were placed in this fixative immediately after injection.

21. Bost, T. C.: Mesenteric Injuries and Intestinal Viability, Ann. Surg. **89**:218, 1929.

22. Rothschild, N. S.: Safety Factors in Mesenteric Ligations, Ann. Surg. **89**:878, 1929.

23. Davies, D. V., and Edwards, D. A.: The Blood Supply of the Synovial Membrane and Intra-Articular Structures, Ann. Roy. Coll. Surgeons of England, 1948, pp. 142-156.

The specimens were then cleared for study in the manner described by Noer [1] and by Lathrop and Krupp.[24]

All the earlier injections were made at pressures between 200 and 250 mm. of mercury. These have since been repeated at pressures of 100 to 150 mm. to approximate more nearly normal arterial pressures. While the vascular filling has been less rapid, the degree and comparative rapidity of filling have shown no significant variations from those obtained at higher pressures.

A total of fifty-one intestinal loops from four different species have been injected after various types of mesenteric vascular interruption, the distribution being as follows:

| | |
|---|---|
| Man | 9 loops |
| Dog | 20 loops |
| Rabbit | 15 loops |
| Opossum | 7 loops |

All loops were chosen from the lower part of the jejunum or upper part of the ileum; more proximal or distal areas were not included so as to avoid anastomoses possibly derived from the gastroduodenal or ileocolic arteries.

The human specimens were obtained at autopsy, in most cases several hours after death. Despite the fact that one might expect difficulty from intravascular clotting and from degenerative changes in the vessel walls, good injections were obtained. In no case did extravasation of the injection mediums occur to a degree incompatible with accurate observation, while the fresher material showed no extravasation whatever. The animals studied were anesthetized by the intravenous injection of pentobarbital sodium and the injections carried on during life; death of the animal usually occurred shortly after completion of the injection.

The dog was chosen for study because of its almost universal use in experimental work, despite its differences from man. The opossum and rabbit were selected because of their easy availability as experimental animals with mesenteric vascular patterns morphologically comparable to that of man.[1]

Three types of mesenteric vascular interruption have been carried out in each of the species mentioned (fig. 1): (1) ligation and division of the intestinal arteries alone to 15 cm. loops, (2) ligation and division of the intestinal arteries to 15 cm. loops plus the arcuate vessels at the extremities of these loops and (3) ligation and division as in (2) with the added interruption of all the vasa recta to the segment. The last two types of ligation have been carried out in some loops more than 15 cm. long for comparison.

24. Lathrop, G. E., and Krupp, R.: Microscopy of Latex Rubber-Injected Specimens, Am. J. Clin. Path. **14**:128, 1944.

In the earlier injection studies some difficulty was experienced in following the sequence of filling of the various vascular elements because of the rapidity with which the vessels filled, even at pressures approximating the normal. This difficulty was finally overcome by "slow motion" cinephotography. Close-up motion pictures of the

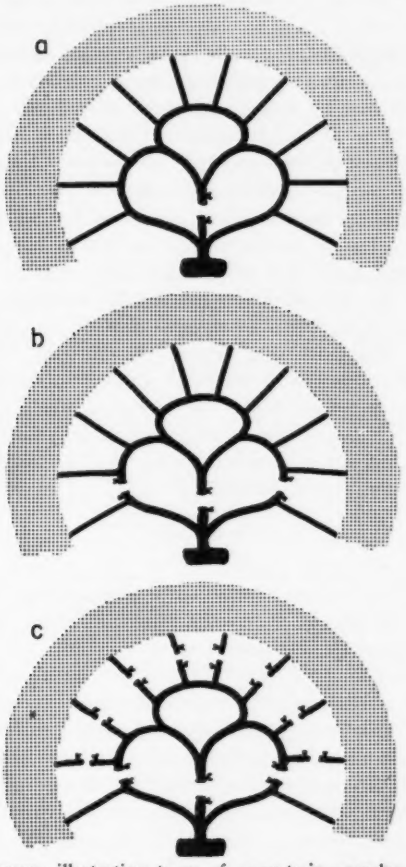


Fig. 1.—Diagrams illustrating types of mesenteric vascular interruption. *a*, division of the intestinal artery alone; *b*, division of the intestinal artery together with the arcuate vessels at each extremity of the loop; *c*, division of the intestinal artery, the arcuate vessels at each extremity of the loop and all the intervening vasa recta.

loops were taken at 64 or 32 frames per second during injection and the developed pictures projected at 16 frames per second to permit detailed study of the course taken by the injection mediums.

## RESULTS

1. *Division of the Intestinal Arteries Alone.*—In every case the injection medium filled the loop rapidly and without difficulty, passing from adjacent intestinal arteries by way of the arcuate vessels and vasa recta to the intestinal wall.

2. *Division of the Intestinal Artery Together with the Arcuate Vessels at Each Extremity of the Loop.*—Vascular filling took place by way of the vessels within the intestinal wall at each extremity of the ligated loop, thence to the arcuate system and vasa recta of the interrupted area and into the mural vessels and their areas of anastomosis. Loops longer than 15 cm. failed to fill completely, injection taking place at the ends, while the midportion of the loops remained clear.

3. *Division of the Intestinal Arteries and Arcuate Vessels Plus All the Vasa Recta to the Loop in Question.*—Good filling was obtained in every case by way of the intramural circulation alone. These specimens filled surprisingly well in every instance, and the rate was only slightly slower than that which occurred when the connections between the arcuate vessels and the loop remained intact. It should be pointed out, however, that there is a definite though brief time lag in the filling of the mural vessels as compared with the arcuate system. It is of interest that the rapidity and the degree of filling obtained in the dog was always less than that in the other three species. Only in this animal did we find some specimens failing to fill completely the central portion of the deprived 15 cm. loops.

## COMMENT

The results following interruption of the intestinal arteries alone are in accord with the usual ideas on the compensatory mechanisms of the intestinal circulation. The results in the second and third types of occlusion, by contrast, show a surprising and unexpected ability of the intramural anastomoses to reestablish circulation in intestinal loops deprived of part or all of their mesenteric vessels. This finding is at variance with most published ideas on the subject, though the possibility was suggested by Welch and Mall in 1897.[13] Since the findings have been repeatedly confirmed in all the species studied, it its apparent that the intramural circulation is of far greater importance in revascularization than has hitherto been supposed.

Several questions are raised by the slower and less complete filling of the dog loops. One of these is the part played by the circular muscular contraction commonly seen in the intestine of this animal in response to vascular deprivation (an effect seldom seen in other species). Many authors have reported this rather striking phenomenon and believe that revascularization is but poorly accomplished in

its presence. We found this contraction the rule in dog intestine, and we know from quartz rod transillumination studies that there is considerable interference with blood flow in the intestinal wall during peristaltic contraction. To eliminate this as a possible factor, 1 dog was killed the day before injection of its intestine. There was no demonstrable difference in the filling of this dead animal's vessels despite complete absence of intestinal contraction.

Another possible explanation for the poorer filling of the dog loops appears to be the type of anastomosis between the vessels within the intestinal wall. Though descriptions of these vessels were included in the 1943 report previously mentioned,[1] it seemed advisable to reevaluate this situation with the present problem in mind. Accordingly the mural circulation was restudied in injected specimens of intestines of man, dog, rabbit and opossum, with the aid of gross inspection and

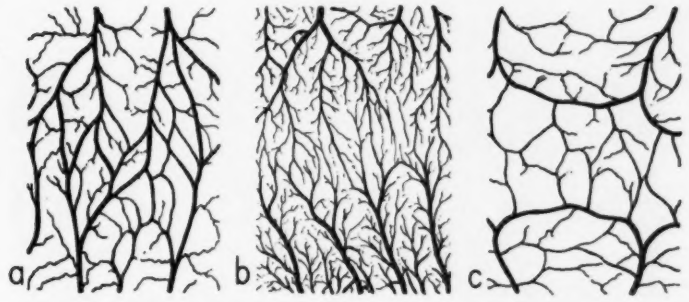


Fig. 2.—Diagram of the type of intramural and antemesenteric anastomoses within the intestinal wall. *a*, direct intercommunications between the mural trunks typically seen in man and the opossum and to a lesser extent in the rabbit; *b*, the "plexiform" anastomotic pattern commonly seen in the dog. Note that the anastomoses are only by fine communicating vessels; *c*, intramural anastomoses formed by arcuate vessels lying near the antemesenteric border. This type is characteristic of the rabbit.

examination with the wide field binocular microscope. These studies confirmed the 1943 report in every respect (figs. 2 and 3). Briefly, the vessels of the intestinal wall may be said to communicate with each other through three general types of anastomoses: (1) direct cross communications between the mural trunks, (2) "plexiform" anastomoses in which the adjacent vessels break up into fine radicles which then anastomose with each other and (3) arcuate communicating vessels which run parallel to the long axis of the intestine to connect adjacent mural vessels along the side of the intestine, usually near the antemesenteric border. The first type is best seen in the human intestine and also in the opossum. The third type is characteristic of the rabbit

and is occasionally seen in the opossum and more rarely in other species. The dog shows chiefly the plexiform type of mural intercommunications and has few direct anastomoses between the mural ves-

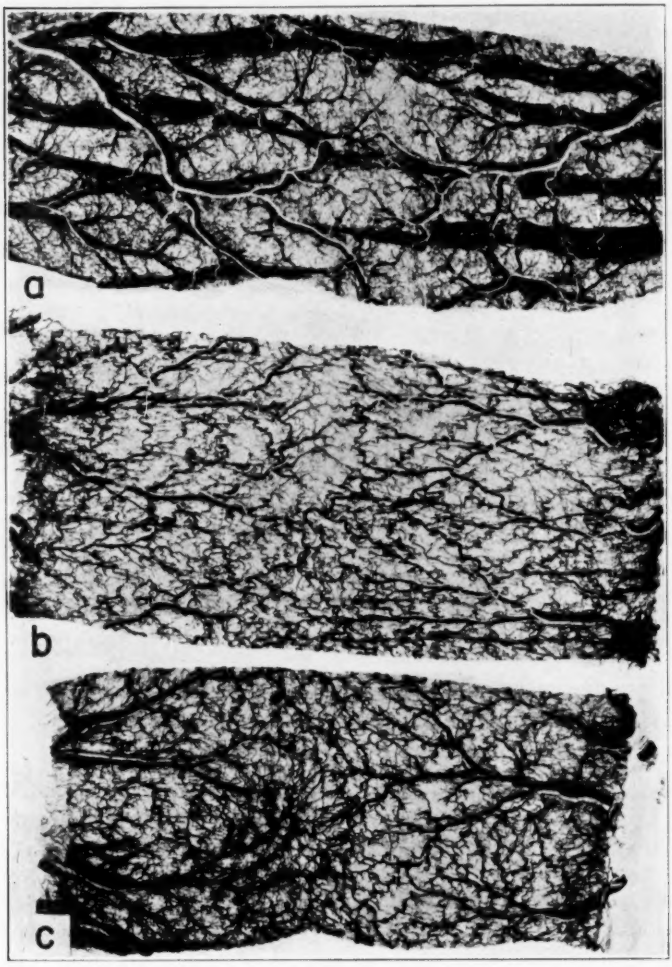


Fig. 3.—Photograph of the anastomoses within the wall and in the antemesenteric border of human specimens (compare with fig. 2). *a*, jejunum; *b*, upper ileum; *c*, lower ileum.

sels. It is our impression that this situation best explains the poorer filling seen in dog loops injected after mesenteric vascular deprivation.

Since the intramural circulation appears to be such an important factor in revascularization, one cannot but speculate on the effect which distention might exert on the efficiency of these anastomoses. Preliminary experiments indicate that even moderate degrees of dis-

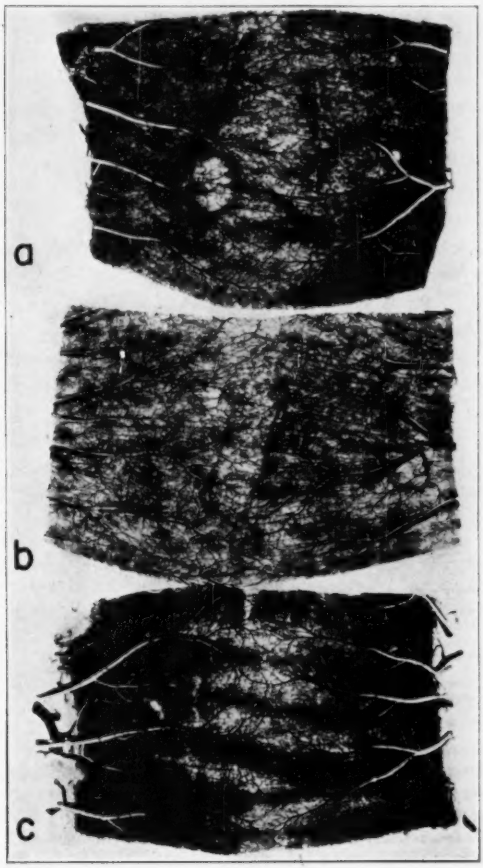


Fig. 4.—Photograph of the mural anastomoses and antemesenteric areas of dog intestine (compare with fig. 2). *a*, jejunum; *b*, upper part of ileum; *c*, lower part of ileum.

tention may greatly vary the ability of the mural circulation to establish blood supply in partially devascularized intestine. If such actually is the case, the need for decompression in the treatment of all types of intestinal distention assumes added importance. Further studies on this point are in progress.

## CONCLUSIONS

1. Injection of partially devascularized intestinal loops provides a convenient method for studying routes available for revascularization. There is sufficient correlation between survival in animals and the ability to inject such loops to justify guarded inferences based on results of injection studies.

2. After ligation of intestinal arteries alone, the deprived loops fill rapidly from adjacent intestinal arteries by way of the arcuate vessels and their vasa recta into the intestinal wall.

3. After ligation of the intestinal arteries together with the arcuate vessels at each extremity of 15 cm. loops of intestine, good filling takes place through the vessels of the intestinal wall, thence to the arcuate system and its vasa recta and into the mural vessels.

4. A 15 cm. loop can be well injected after ligation of its intestinal arteries, the arcuate vessels at each extremity and all its vasa recta. In this case filling is accomplished solely by anastomoses between the vessels of the intestinal wall.

5. The revascularizing potential of the dog in these circumstances appears to be somewhat less than that of man, opossum and rabbit.

6. While its efficiency in a given species appears to be dependent on the type of intramural anastomoses, it is clear that the circulatory system within the intestinal wall provides an added and important compensatory mechanism over and above that found in the arcuate system.

Dr. Charles G. Johnston, Professor of Surgery, and Dr. Gordon H. Scott, Professor of Anatomy, Wayne University College of Medicine, gave their continued interest and helpful suggestions during the course of this investigation.

## DISCUSSION

DR. CHARLES G. JOHNSTON, Detroit: I have little to say except that I hate to miss this opportunity to say a word about this rather painstaking work which Dr. Noer and Dr. Derr have been carrying on for several years. I am sure those of you who do any amount of intestinal surgery will get a great deal of comfort from the pictures illustrating the amount of revascularization that is possible through the intestinal wall. I have long feared the use of a loop which has been fairly well devascularized, especially in the use of small intestine for the Roux Y procedure, always fearing that perhaps I was taking off too much mesentery. It is pretty obvious from these experiments that so long as one maintains the arcuate system there is a good anastomotic blood flow through the entire area of the gut, so one can take a fairly long loop. The great trouble usually is that the mesentery holds the small bowel down, and it becomes necessary to free it. These studies give me a great deal of comfort.

Dr. Noer and Dr. Derr have been carrying on these studies in a large series of species, and the great amount of detail which they have been able to bring out in the vessels in the wall itself is amazing. I must say that I was critical of

their work when I first saw them, because I had no idea that so fine an anastomosis could be obtained through the intestinal wall itself, with all the arcuate vessels completely tied. This is a long loop to devascularize completely and still maintain a blood supply. Dr. Derr and Dr. Noer have not only done this so far as the injected specimens are concerned but likewise have done it in several species of animals, and the results are the same. I am sure that from their comparable results on autopsy material from human beings we can expect a much better vascularity in the intestinal tract than we had previously supposed existed; so we have a great deal of leeway in the use of intestine that is partially devitalized.

Dr. John H. Olwin, Chicago: Is there any information as to the ease of the return circulation compared to the arterial flow in the animals that survived? Was there any noticeable engorgement of the loops in the animals that survived?

Dr. Rudolf J. Noer, Detroit: There was no engorgement of the loops in the animals that survived, with the exception of those in which only the veins were ligated. There was some engorgement when the veins were ligated without the arteries.

As far as the ease of return of the circulation is concerned, those animals which had an arterial deprivation alone appeared somewhat pale immediately after the ligation. The loops were not kept out of the abdomen very long, and on examination at autopsy after ten day survivals the loops appeared somewhat anemic but otherwise were relatively normal. They were rather pale in appearance and somewhat smaller in caliber than the other loops.

# USE OF PORTACAVAL ANASTOMOSIS IN PORTAL CIRRHOSIS

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PORTAL cirrhosis is progressively increasing in importance as a medical and economic problem. The rising incidence of this disease at autopsy at the Los Angeles County Hospital is indicated in table 1. The reports of Whipple,[1] Blakemore[2] and Blalock[3] on portacaval anastomosis have established the feasibility of surgical alleviation of certain of the distressing features exhibited by patients with advanced portal cirrhosis. The experience of my colleagues and me with the problem is presented in the following report of 14 cases, in 10 of which this operative procedure has been carried out. In all 10 cases the portal vein has been anastomosed to the inferior vena cava.

The portal system of veins is interposed between two capillary beds, that of the gastrointestinal tract, pancreas, spleen and gallbladder and that of the liver. Obstruction involving the capillaries of the bed of the liver or of the major vessels of the portal system may produce portal hypertension. Therefore, cases of portal hypertension can be divided into two main groups, those with intrahepatic portal block and those with extrahepatic portal block.

Cirrhosis of the liver, particularly the portal type, is by far the most common cause of intrahepatic portal block. Early in portal cirrhosis there are few if any clinical symptoms or signs, and often it is not until the resulting portal hypertension becomes excessive that there is awareness of the disease. Also, in patients with portal cirrhosis of pathologically similar degree the clinical manifestations vary. Thus, 1 patient

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From the Department of Surgery, University of Southern California, Los Angeles.

Read at the Fifty-Sixth Annual Meeting of the Western Surgical Association, St. Louis, Dec. 2, 1948.

1. Whipple, A. O.: The Problem of Portal Hypertension in Relation to the Hepatosplenopathies, Ann. Surg. **122**:449 (Oct.) 1945; The Rationale of Portacaval Anastomosis, Bull. New York Acad. Med. **22**:251 (May) 1946.

2. Blakemore, A. H.: Portacaval Anastomosis: A Report of Fourteen Cases, Bull. New York Acad. Med. **22**:254 (May) 1946; Indications for Portacaval Anastomosis-Analysis of Cases, Surg., Gynec. & Obst. **84**:645 (April) 1947.

3. Blalock, A.: The Use of Shunt or By-Pass Operations in the Treatment of Certain Circulatory Disorders, Including Portal Hypertension and Pulmonary Stenosis, Ann. Surg. **125**:129 (Feb.) 1947.

may have no signs of excessive portal pressure, another may have splenomegaly, another splenomegaly with hematemesis, another ascites only and others ascites with splenomegaly alone or associated with hemorrhages. To date our surgical experience has been confined entirely to the group presenting either ascites and/or hemorrhages from esophageal varices.

The surgical approaches of the past, for the most part, have proved disappointing in alleviation of both morbidity and mortality in this dis-

TABLE 1.—*Incidence of Portal Cirrhosis at Autopsy at Los Angeles County Hospital*

| Year | Number of Cases | Cases of Portal Cirrhosis | Percentage |
|---|---|---|---|
| 1918 to 1932 | 9,527 | 62 | 0.65 |
| 1933 to 1937 | 8,347 | 155 | 1.84 |
| 1938 | ..... | .. | .... |
| 1939 | 2,097 | 73 | 3.48 |
| 1940 | 2,308 | 91 | 3.94 |
| 1941 | 2,185 | 130 | 5.95 |
| 1942 | 2,109 | 122 | 5.78 |
| 1943 | 1,801 | 90 | 4.99 |
| 1944 | 1,638 | 71 | 4.34 |
| 1945 | 1,527 | 87 | 5.69 |
| 1946 | 1,785 | 116 | 6.49 |
| Total | 33,324 | 997 | |

TABLE 2.*—*Prognosis in Portal Cirrhosis with Ascites*

| | Mortality | |
|---|---|---|
| | Treated Patients (115), Percentage | Untreated Patients (230), Percentage |
| 1 yr | 35 | 61 |
| 2 yr | 50 | 79 |
| 5 yr | 70 | 93 |

* From the article by Patek and others.[5]

ease. Therefore, before considering any operative procedure, it would be well first to analyze the prognosis in portal cirrhosis. Ratnoff and Patek [4] and Patek and his associates [5] have made such a study. Table 2 is adapted from the latter and illustrates the outlook in cases with ascites. At the present time there are no adequate figures for the group of patients presenting hemorrhage from esophageal varices who have

4. Ratnoff, O. D., and Patek, A. J.: The Natural History of Laennec's Cirrhosis of the Liver, Medicine **21**:207 (Sept.) 1942.

5. Patek, A. J.; Post, J.; Ratnoff, O. D.; Mankin, H., and Hillman, R. W.: Hepatic Cirrhosis, J. A. M. A. **138**:543 (Oct. 23) 1948.

had the obvious benefit of a present day medical regimen. However, previous figures [4] on untreated series show the group with hematemesis to have a mortality curve similar to the group with ascites. Also, it is most interesting, and certainly significant therapeutically, that 40 per cent of patients who have hemorrhage from esophageal varices die within one month of the first hemorrhage.

All this crystallizes into an ominous prognosis for the person with advanced portal cirrhosis, and it would seem that any operative procedure, provided it will give a significant measure of relief to the patient, is worthy of serious consideration. Decreasing the portal hypertension by portacaval anastomosis should, on a purely mechanical basis, greatly lower the incidence of hemorrhage. With the ascites of portal cirrhosis resulting from an increase in portal hydrostatic pressure in conjunction with a reduced osmotic pressure of the blood and probably an increase in capillary permeability, measures aimed at decreasing the portal pressure also should aid in the control of the ascites. What the decrease in portal pressure and blood flow does to the functional status of the liver is still conjectural, although, as will be shown subsequently, there is no evidence that shunting the portal blood away from the cirrhotic liver decreases its function.

Table 3 is a fairly comprehensive list of the surgical procedures that have been employed in portal cirrhosis. Until recently none of these procedures has carried any significant degree of promise, and in large series reported [6] 40 to 55 per cent of the patients operated on were dead within one month. The surgical procedures most frequently utilized in the past were (1) splenectomy, (2) ligation of the tributaries to the esophageal varices, (3) omentopexy and (4) peritoneosubcutaneous drainage. None of these procedures has produced sufficiently satisfactory results. On the basis of the vascular physiology in portal cirrhosis as it is now known and with the development of satisfactory methods of vascular anastomosis, the shunting of the portal blood stream into the inferior vena cava appears to be a logical procedure, not with the thought of altering the already developed hepatic changes, but with the hope of controlling ascites and/or esophageal hemorrhages. Of the two basic operative procedures, anastomosis of the splenic vein to the left renal vein or anastomosis of the proximal end of the severed portal vein to the inferior vena cava, we feel that the latter is the preferable operation in portal cirrhosis for two reasons: (1) the larger stoma will conduct more blood and consequently lower the portal pressure to a greater degree, and (2) the animal experiments of Blalock [3] indicate a higher incidence of postoperative thrombosis at the site of splenorenal anastomoses.

6. Henrikson, E. C.: Cirrhosis of the Liver, Arch. Surg. **32**:413 (March) 1936.

Fourteen patients with portal cirrhosis, presenting either ascites or hemorrhage from esophageal varices, have been operated on, and an end to side portal vein—inferior vena cava anastomosis has been accomplished in 10 cases. In 1 of the 4 cases in which the anastomosis was not done a dense plastic peritonitis, following over seventy paracenteses, made identification of any intra-abdominal structure almost impossible. In another case, with hemorrhage from esophageal varices, in which the spleen had been removed four years previously, there was completely organized thrombotic obliteration of the portal vein. Another patient with severe ascites also had completely organized thrombotic obliteration

TABLE 3.—*Operations for Portal Cirrhosis (Portal Hypertension)*

| |
|---|
| A. Establishing Collateral Circulation |
| 1. Omentopexy |
| Morison-Talma—G. O. to anterior abdominal wall plus glass tube |
| Turner—G. O. between liver and diaphragm |
| Schiassi-Barker—G. O. between peritoneum and posterior rectus |
| Narath—G. O. between skin and superficial fascia |
| 2. Visceropexy |
| Hepatopexy |
| Splenopexy |
| To peritoneum (Bunge) |
| To pockets in abdominal wall |
| Testicle and venous plexus intra-abdominally to G. O. (Lanz) |
| Excision of patches of peritoneum |
| B. Ligation of Vessels |
| 1. All tributaries of portal vein have been ligated, except splenic |
| Coronary |
| Inferior mesenteric (Moynihan) |
| Superior mesenteric |
| 2. Ligation of splenic artery |
| 3. Injection of esophageal varices (Moersch) |
| 4. Gastric resection (Wangensteen) |
| 5. Esophagogastrectomy (Phemister—for extrahepatic obstruction) |
| C. Short Circuiting Operations |
| 1. Eck Fistula (Vidal—1906) |
| 2. Superior mesenteric to spermatic or ovarian veins |
| 3. Superior mesenteric to inferior vena cava (Bogoraz and Krestovsky) |
| D. Methods for Draining Ascitic Fluid |
| 1. Saphenoperitoneal anastomosis (Ruotte) |
| 2. Subcutaneous drainage through femoral canal (Handley) |
| 3. Peritoneosubcutaneous drainage with glass tubes (Bocarro-Tannahill) |
| 4. Bladder fistula |
| 5. Pyeloperitoneal anastomosis |
| E. Splenectomy |

of the portal vein. This man died two months after the exploration, just prior to operation for splenorenal shunt, death being the result of an initial massive hemorrhage from esophageal varices. Operation on the fourth patient, a man with severe ascites, had to be terminated because of excessive hemorrhage from the veins of Retzius, encountered while the duodenum and head of pancreas were being elevated from the inferior vena cava. This man received 4,500 cc. of blood but succumbed to hemorrhagic shock postoperatively.

In 10 cases an end to side portal vein—inferior vena cava anastomosis was completed. Death occurred postoperatively in 1 case (8) and was the result of hemorrhage from the inadvertent tearing of the

pyloric vein at its junction with the portal vein, the hemorrhage occurring postoperatively. All the other patients survived the operation and were discharged ambulatory from the hospital. Table 4 lists the operative findings, other than the cirrhosis, that are considered important from a prognostic standpoint. In 4 cases, 1, 5, 8 and 9, the essential disability was massive hemorrhage from esophageal varices, and in the remainder the problem was medically uncontrollable ascites. All patients had been on medical management for prolonged periods. The relative size of the spleen is indicated in the table, as are the pressures in the portal vein and inferior vena cava in centimeters of water. On the basis of this small series there are three operative findings that seem to be significant in the prognostication of the ultimate result. These are the size of the spleen, the height of the pressure in the portal vein

TABLE 4.—*Venous Pressure at Operation (Normal Pressure in Portal Vein, 6 to 12 Cm. of Water)*

| Case | Preshunt | | Postshunt | Size of Spleen |
|---|---|---|---|---|
| | Portal Vein | Vena Cava | Portal Vein | |
| 1 * | 35 | 15 | .. | 5 times normal |
| 2 † | 32 | 20 | .. | 3 times normal |
| 3 † | 12 | 5 | 9 | Normal |
| 4 † | 28 | 18 | 22 | 4 times normal |
| 5 * | 60 | 12 | 26 | 8 times normal |
| 6 † | 35 | 9 | 28 | 8 times normal |
| 7 † | 35 | 15 | 23 | 3 times normal |
| 8 * | 33 | 14 | 17 | Absent |
| 9 * | 40 | 14 | 17 | 8 times normal |
| 10 † | 25 | .. | 20 | 1.5 times normal |

* Hemorrhage from esophageal varices occurred in these cases.
† Ascites was present in these cases.

and the difference in the degree of pressure between the portal vein and the inferior vena cava. In case 6 the expressed criteria would prognosticate a good end result. Unfortunately an organized portal vein thrombosis which had recanalized was found at operation as an additional finding, the lumen of the vein being approximately one-third the normal size. In spite of this an anastomosis was done, with a resulting drop of 7 cm. of water in the pressure of the portal vein. This was enough to lengthen the period between abdominal paracenteses from less than one week to a ten to fourteen day interval. Possibly a splenorenal anastomosis would have been preferable in this case, and it was being considered as a future procedure until the sudden death of the patient at home six weeks postoperatively from massive hemorrhage of the upper intestinal tract.

Several years must elapse before one can assess the true value of this operative procedure. Table 5 summarizes the results obtained

in this series to date. The results in the group with massive hemorrhage from esophageal varices have been particularly gratifying, for no subsequent massive hemorrhages have occurred. The regression in the size of the varicose veins from the decrease in pressure in the portal vein might be compared to the decrease in the size of varicosities in the lower extremities following reduction in pressure from ligation of the saphenous vein.

The group with ascites is more complex, mainly because of the multiple factors involved in the production of ascites. In only 1 case, 4, was the ascites immediately controlled after operation. In case 3, in which no postoperative improvement was shown, the anastomosis should not have been done because studies of venous pressure failed to show portal hypertension. One fact seems to be developing, that assessment of the end result cannot be made immediately. In case 2

TABLE 5.—*End Results of Portacaval Anastomoses*

| | |
|---|---|
| **Group with Hemorrhage** | |
| 1 | No further hemorrhages |
| 5 | No further hemorrhages |
| 9 | No further hemorrhages |
| 8 | Postoperative death—hemorrhage |
| **Group with Ascites** | |
| 2 | No ascites |
| 4 | No ascites |
| 3 | No improvement |
| 10 | No improvement |
| 7 | Moderate improvement |
| 6 | Slight improvement; sudden death six weeks after leaving hospital from massive hemorrhage of upper part of intestinal tract |

there was ino improvement whatsoever for five months, then a gradual lessening of the ascites over a two month period, and in the past six months there has been no evidence of intra-abdominal fluid. This same mechanism seems to be working in cases 7 and 10.

The most grave objection to this operation has been the conjecture relative to the functional status of the already damaged liver when the portal blood flow is completely shunted from its parenchyma. In reality this objection was answered by McIndoe[7] in 1928, when he demonstrated by infusion studies how really little of the portal blood flow passes directly through the cirrhotic liver. In essence, the patient with portal cirrhosis attempts to establish his own Eck fistula through the medium of the extensive collateral circulation that develops.

Many preoperative and postoperative liver function tests have been done in all these cases. Table 6 lists the highest serum albumin and

7. McIndoe, A. H.: Vascular Lesions of Portal Cirrhosis, Arch. Path. **5**:23 (Jan.) 1928.

globulin measurements before and after shunt. In no instance was there a significant drop in the serum albumin postoperatively, and in the first 2 patients operated on there is now an appreciable rise. The cephalin flocculation, thymol turbidity and sulfobromophthalein tests uniformly have shown no decrease in function and often a significant improvement. In this series the prothrombin time has been neither normal nor down to a critical level preoperatively or postoperatively, with one exception. In case 1, between the second and third postoperative weeks, a considerable amount of blood oozed from the gastrointestinal tract. At this time all reactions to liver function tests were normal except for the prothrombin time, which was in the bleeding range. Sixty milligrams of vitamin K daily was required to keep the prothrombin time above the bleeding range. This dosage was slowly

TABLE 6.—*Serum Albumin and Globulin Levels in Portal Cirrhosis*

| Case | Preshunt | | Postshunt | | Interval, Mo. |
|---|---|---|---|---|---|
| | Albumin | Globulin | Albumin | Globulin | |
| 1 | 4.4 | 3.9 | 4.5 | 2.2 | 15 |
| 2 | 3.4 | 3.6 | 5.1 | 2.7 | 13 |
| 3 | 4.0 | 2.8 | 3.8 | 2.7 | 10 |
| 4 | 4.0 | 4.7 | 3.8 | 4.2 | 11 |
| 5 | 3.8 | 1.4 | 4.0 | 1.2 | 10 |
| 6 | 2.6 | 1.0 | 2.6 | 2.6 | 1 |
| 7 | 3.7 | 3.0 | 3.5 | 2.2 | 6 |
| 8 | 3.5 | 2.9 | ... | ... | .. |
| 9 | 3.2 | 2.7 | 3.2 | 2.8 | 2 |
| 10 | 3.6 | 2.9 | 2.8 | 2.9 | 2 |

decreased until the end of the second postoperative month, when 12 mg. daily was adequate. This dosage had to be maintained until the fifteenth postoperative month, the prothrombin time from then on remaining at a normal level without the use of vitamin K.

## SUMMARY OF CASES

CASE 1.—A white woman, aged 46, with chronic alcoholism, had repeated severe hemorrhages of the upper part of the gastrointestinal tract for fourteen months, being hospitalized for a total of twelve months during this period. On physical examination the edge of the liver was 5 cm. below the costal margin, there was no ascites and the spleen was not palpable. Liver function tests revealed depression following hemorrhages, but all tests gave results which were within normal limits preoperatively. The serum albumin and globulin levels were 4.4 and 3.9 Gm. per hundred cubic centimeters respectively. At operation on March 7, 1947, the liver was moderately enlarged, firm and nodular, and the biopsy revealed moderate portal cirrhosis. There was no ascites, and the spleen was estimated at five times the normal size. The pressure in

the portal vein was 35 cm. of water, and that in the inferior vena cava was 15 cm. of water. Postoperatively, during the second week, oozing developed in the gastrointestinal tract, the prothrombin time was 5 per cent of normal and the patient had to be given 60 mg. of vitamin K daily to keep the prothrombin time above the bleeding range. The necessary dosage slowly decreased, although vitamin K had to be given daily for fifteen months. During this period all other liver function tests gave normal results. The present serum albumin and globulin levels are 4.8 and 2.2 Gm. respectively. The patient has been working since the fourth postoperative month and has had no subsequent hemorrhages.

Case 2.—A Mexican woman, aged 40, with chronic alcoholism, had pronounced ascites continuously for six months which necessitated weekly paracentesis. On physical examination the liver was not palpable, there was marked ascites and the spleen was not palpable. Liver function tests showed moderate depression, the initial serum albumin and globulin levels being 2.9 and 3.9 Gm. per hundred cubic centimeters and ultimately stabilizing at 3.4 and 3.6 Gm. respectively. At operation on Sept. 5, 1947, the liver was slightly contracted, firm and nodular, and biopsy revealed moderate portal cirrhosis. There was considerable ascites, and the spleen was estimated at three times normal size. The pressure in the portal vein was 32 cm. of water and that in the inferior vena cava 20 cm. of water. Immediately after operation there was no change in the degree of ascites. However, seven months later the ascites had completely regressed, and there has been no subsequent intra-abdominal fluid. The present serum albumin and globulin levels are 5.1 and 2.7 Gm. per hundred cubic centimeters respectively.

Case 3.—A white woman aged 46 had portal cirrhosis of unknown cause. There had been abdominal distention for one and one-half years, which necessitated paracentesis at bimonthly intervals. Physical examination revealed decided ascites, and the liver and spleen were not palpable. After medical treatment the serum albumin and globulin levels were 4.0 and 2.8 Gm. per hundred cubic centimeters respectively. At operation on Sept. 26, 1947, the liver was normal in size, firm and finely nodular. Biopsy revealed portal cirrhosis with regeneration. The spleen was normal in size, and the pressure in the portal vein was 12 cm. of water while that of the inferior vena cava was 5 cm. of water. After anastomosis the pressure in the portal vein was 9 cm. It has been necessary to continue paracentesis at monthly intervals. The serum albumin and globulin levels are now 3.8 and 2.7 Gm. respectively.

Case 4.—A Mexican man, aged 43, with chronic alcoholism, was known to have cirrhosis of five years' duration and had had ascites for one year which necessitated ten paracenteses. On physical examination the liver was 4 cm. below the costal margin, there was notable ascites and the spleen was not palpable. After medical treatment the serum albumin and globulin levels were 4.0 and 4.7 Gm. per hundred cubic centimeters respectively. At operation on Oct. 12, 1947, the liver was moderately decreased in size, firm and hobnailed, and biopsy revealed advanced portal cirrhosis. The spleen was four times normal size, the pressure in the portal vein was 28 cm. of water and that in the inferior vena cava was 18 cm. of water. After anastomosis the pressure in the portal vein was 22 cm. At no time has this patient shown evidence of abdominal fluid since operation. The serum albumin and globulin levels are now 3.8 and 4.2 Gm. respectively.

CASE 5.—A white man, aged 47, had four severe hemorrhages in the ten months preceding operation. The liver was not palpable, there was no ascites and the spleen was palpable 6 cm. below the costal margin. All liver function tests gave essentially normal results immediately after operation. The serum albumin and globulin levels were 3.8 and 1.4 Gm. per hundred cubic centimeters respectively. Roentgen study revealed extensive varicosities extending up into the cervical esophagus. At operation on Oct. 23, 1947, the liver was of normal size and finely nodular, and biopsy revealed portal cirrhosis. There was no ascites, and the spleen was at least eight times the normal size. The pressure in the portal vein was 60 cm. of water, and the pressure in the inferior vena cava was 12 cm. of water. After anastomosis the pressure in the portal vein was 26 cm. On the patient's discharge, seven days postoperatively, the spleen was no longer palpable. The subsequent serum albumin and globulin levels measured 4.0 and 1.2 Gm. per hundred cubic centimeters respectively. There have been no further hemorrhages, and the patient's general health is excellent.

CASE 6.—A white woman, aged 40, became ill seven years previous to her admission to the hospital with hematemesis, tarry stools and ascites. She was operated on four years previously, when the splenic vessels were ligated. Subsequently there had been mild hemorrhages on three occasions and a progressively severe ascites, which in the past two months had necessitated paracentesis at seven day intervals. On physical examination the liver was not palpable, there was considerable ascites and the spleen was 6 cm. below the costal margin. Roentgen study revealed the presence of moderate esophageal varices. All liver function tests showed considerable depression, and after medical treatment the serum albumin and globulin levels were 2.6 and 1.0 Gm. per hundred cubic centimeters respectively. At operation on Jan. 10, 1948, the liver was markedly contracted, firm and hobnailed, being less than one third the normal size. There was pronounced ascites, and the spleen was eight times the normal size. The portal vein was two thirds occluded by an old thrombus which was completely organized. The pressure in the portal vein was 35 cm. of water, and that in the inferior vena cava was 9 cm. of water. After anastomosis the pressure in the portal vein was 28 cm. Postoperatively the interval between paracenteses lengthened to ten to twelve days. The serum albumin and globulin were 2.6 and 2.6 Gm. per hundred cubic centimeters respectively. The patient died at home forty-three days postoperatively from a massive hemorrhage in the upper gastrointestinal tract. At atopsy the site of anastomosis was completely occluded by a recent thrombus.

CASE 7.—A white man, aged 50, with chronic alcoholism, had intermittent ascites for three years and continuous ascites for one year. An unsuccessful peritoneosubcutaneous drainage had been done seven months previously. In the past year paracentesis had been performed twenty-six times, with a total of 668 pounds (303 Kg.) of fluid removed, the protein content of which was estimated at forty times the amount of the normal circulating albumin. Physical examination revealed ascites and an umbilical hernia; the edge of the liver was 5 cm. below the costal margin, and the spleen was 2 cm. below the costal margin. After medical treatment the serum albumin and globulin levels were 3.7 and 3.0 Gm. per hundred cubic centimeters respectively. At operation on Jan. 19, 1948, the liver was contracted, firm and nodular, and biopsy revealed portal cirrhosis. The spleen was estimated at three times the normal size, the pressure

in the portal vein was 35 cm. of water and that in the inferior vena cava was 15 cm. of water. After anastomosis the pressure in the portal vein was 23 cm. At the present time paracentesis is necessary at monthly intervals. His average gain in weight is now 0.7 pounds (0.35 Kg.) per day whereas before operation it was 3.0 pounds (1.3 Kg.) per day. The present serum albumin and globulin levels are 3.5 and 2.2 Gm. respectively.

Case 8.—A white male aged 25 had his first massive hemorrhage in the upper gastrointestinal tract in 1943. In 1944 a splenectomy was done, the pathologic report being "fibrosis of the spleen—Banti's syndrome." In 1946 there was a short interval of jaundice. In the first six months of 1948 he had two severe hemorrhages of the upper intestinal tract. On physical examination there was a scar in the upper midline area, the edge of the liver was just palpable and there was no ascites. Roentgen study revealed the presence of moderate esophageal varices. After medical treatment the serum albumin and globulin levels were 3.5 and 2.9 Gm. per hundred cubic centimeters respectively. At operation on July 10, 1948, the liver was not enlarged and was firm and finely nodular; biopsy revealed portal cirrhosis. The spleen was absent, and no accessory spleens were found. The pressure in the portal vein was 33 cm. of water and that in the inferior vena cava 14 cm. of water. An old organized thrombus was present in the portal vein, which only slightly occluded the lumen. After anastomosis the pressure in the portal vein was 17 cm. of water. The patient died nine hours postoperatively from shock secondary to massive intra-abdominal hemorrhage. At autopsy the source of the hemorrhage was the portal vein at the site where the pyloric vein had been torn across. The anastomosis was patent.

Case 9.—A white woman aged 54 had been ill for twenty-four months, during which time she had six massive gastorintestinal hemorrhages. On physical examination the edge of the liver was down 3 cm. below the costal margin, there was no ascites and the spleen was down 6 cm. below the costal margin. Roentgenologic study revealed the presence of moderate esophageal varices. After medical treatment the serum albumin and globulin levels were 3.2 and 2.7 Gm. per hundred cubic centimeters respectively. At operation on Aug. 10, 1948, the liver was of normal size, firm and finely nodular; biopsy revealed portal cirrhosis. There was no fluid, and the speen was eight to ten times the normal size. The pressure in the portal vein was 40 cm. of water, and that in the inferior vena cava was 14 cm. of water. After anastomosis the pressure in the portal vein was 17 cm. On the fourth postoperative day the spleen was no longer palpable. There have been no subsequent hemorrhages. The serum albumin and globulin levels are now 3.2 and 2.8 Gm. respectively.

Case 10.—A white man aged 26, whose cirrhosis was of undertermined origin, although possibly he had had infectious hepatitis three years previously, had ascites for twenty-eight months, necessitating aspiration at four week intervals. On physical examination there was ascites, the edge of the liver was 4 cm. below the costal margin and the spleen was not palpable. After medical treatment the serum albumin and globulin levels were 3.5 and 2.9 Gm. per hundred cubic centimeters respectively. At operation on Aug. 27, 1948, the liver was moderately enlarged, firm and hobnailed, and biopsy revealed portal cirrhosis. The spleen was estimated at one and one-half times normal size, and the pressure in the portal vein was 25 cm. of water. After anastomosis this pressure

was 20 cm. of water. Postoperative paracentesis was done at two week intervals. Subsequently this interval has slowly increased, until at the present parecentesis is done at four week intervals. The present serum albumin and globulin levels are 2.8 and 2.9 Gm. respectively.

## CONCLUSIONS

1. The incidence of portal cirrhosis at autopsy is increasing.

2. The poor prognosis in portal cirrhosis warrants the use of any operative procedure that offers a significant measure of relief to the patient.

3. Anastomosing the portal vein to the inferior vena cava shows definite promise in the alleviation of ascites and hemorrhage from esophageal varices in portal cirrhosis.

4. The functional status of the liver in cases of portal cirrhosis, following portacaval anastomosis, is not decreased.

## DISCUSSION

DR. LOUIS G. HERRMANN, Cincinnati: At the Cincinnati General Hospital we have also been interested in the various surgical methods for reducing hypertension in the portal circulation. Dr. Pattison has presented the indications for these surgical procedures, and he has given us an excellent summary of the results which he has obtained.

We have found that the anastomosis of the splenic vein to the renal vein in most patients with portal hypertension has been unsatisfactory. This has been due either to the technical difficulties of getting a long enough splenic vein to reach to the renal vein or, more often, to later thrombosis of the splenic vein at the site of the anastomosis.

In most of our patients the spleen has been found to be densely adherent to the diaphragm; consequently we adopted the thoracoabdominal approach to the spleen which Dr. B. N. Carter described many months ago. The illustration on the screen shows what an excellent exposure is obtained by this incision. The pedicle of the spleen is directly in front of the surgeon, and the adhesions to the diaphragm can be severed under direct vision.

More than a year ago we adapted this thoracic and abdominal incision to the right side in order to obtain an adequate exposure of the portal vein and the inferior vena cava at a point just below the liver. We believe that the portal circulation can be decompressed into the systemic venous circulation more effectively by a shunt between the portal vein and the inferior vena cava. Dr. Pattison has presented evidence that the portal vein can be completely severed and the entire portal circulation diverted into the vena cava without serious consequences. We have chosen to make a side to side anastomosis between the portal vein and the inferior vena cava so as not to divert completely the portal blood from the liver.

There are still many technical obstacles which must be overcome before the major hazards of the surgical treatment of portal hypertension can be considered as minimal. The portal vein has occasionally been found to consist of a group of small venous channels, so the blood could not be diverted into the systemic venous circulation by any surgical procedure.

Adequate exposure of all these structures is of paramount importance, so it is our hope that more surgeons will utilize the right thoracoabdominal approach in performing the anastomosis between the portal vein and the inferior vena cava. The ease with which the exposure can be accomplished should further reduce the hazards of this type of surgery.

Dr. C. J. Berne, Los Angeles: I have had the pleasure of watching Dr. Pattison's work. I think that in his report he had no intention of competing with various other procedures such as "packing" of the esophagus, gastric surgery and other types of portacaval anastomoses. I wish to make one point regarding portacaval anastomosis which he did not have time to cover:

(Slide) This comes close to being the crux of the real reason why complete division of the portal vein and anastomosis to the vena cava is physiologically possible, because the blood so shunted into the cava then reaches the same physiologic unit in the liver via the hepatic artery through the systemic circulation as that blood which has been previously going via the portal system into the sinusoid and in that way arrives at the same functional liver unit.

I believe that this particular point is the basis for all the various operations which have been attempted in creating these shunts. Dr. Pattison has had the privilege of reporting 10 cases of end to side anastomoses. Early, in watching these operations, it was my impression that the magnitude of the spontaneous portacaval anastomoses by the veins of Retzius in the region of the spleen and the left kidney was so great that the removal of these by splenectomy constituted almost as much of a deprivation in collateral as was added by a lienorenal shunt. For that reason the particular procedure which has been carried out was used.

The end to side anastomoses have been done by taking the button out of the vena cava, and I think that a larger connection than would be obtained with the ordinary incision anastomosis has been accomplished.

One of the important problems in diagnosis, left to be solved, has to do with the thrombotic problems incident to cirrhosis of the liver, in which subhepatic thrombosis of the portal vein has occurred in the past. As yet we have not come on any way, historically or otherwise, to recognize that previous thrombosis has occurred and therefore that the so-called cavernous transformation of the portal vein is existent.

The postoperative thrombotic complications, which have been more numerous in the group reported by Dr. Blakemore, fortunately have not yet occurred. I think it is an interesting phase of the problem and possibly is not related to ordinary thrombotic mechanisms but may turn out to have some relation to withdrawal of the splenic function or, rather, of the hypersplenism incident to portal hypertension and the withdrawal of these splenic overactivities and to the relation to the thrombocyte level in the blood during the early postoperative period when these thromboses occur. Studies in regard to this may be an interesting phase of the whole problem, because the secondary thromboses are generally considered to be one of the serious hazards in the operation.

Dr. Arthur C. Pattison, Los Angeles: In reference to Dr. Herrmann's discussion concerning the technical difficulty of this operation, may I make one comment? Our own experience has been that in that group of persons who have hemorrhage from esophageal varices the operation has been infinitely more easily done than in that group with ascites and for essentially two reasons: First, those who have hemorrhage from the esophageal varices do not seem to have the large

veins of Retzius communicating between the duodenum and the head of the pancreas and the liver. Second, those who have ascites have hypoproteinemia or hypoalbuminemia at the time they are operated on, and the edema makes that particular technical maneuver of elevating the duodenum and the head of the pancreas a little more difficult.

I might also stress the fact that, as Dr. Berne said, the establishment of a button, cut from the wall of the inferior vena cava to the size of the circumference of the portal vein, is extremely important for one other reason, and that is that by making a button in the inferior vena cava we feel (although we have no proof for it) that the incidence of thrombosis at the site of the anastomosis will be decreased.

# USE OF HEPARIN AND DICUMAROL IN THE ACTIVE TREATMENT OF THROMBOEMBOLISM

JOHN H. OLWIN, M.D.
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ADVANCE in recent years in the knowledge of the blood coagulation mechanism and methods for its control has been a major stimulus to investigators in the field of thromboembolism. Reports of the reduction of its incidence postoperatively with the prophylactic use of anticoagulants [1] and the lowering of the mortality rates and complications in coronary thrombosis [2] have been encouraging. Once a thrombosis develops, the favorable effect of anticoagulants would appear to be a prevention of the spread of the lesion and of the formation of loose clots easily dislodged from the parent thrombus. In addition, recent work [3] suggests an increase in the arterial blood flow from administration of dicumarol® and, to a less extent, heparin. There is evidence at present to indicate that the newly discovered prothrombin accelerator factor [4] is increased early in the thrombotic process,[5] and two groups of workers have found it to be depressed in the initial stages of dicumarol® therapy.[6] It is the purpose of this paper to report experience with the use of heparin and dicumarol® in the active treatment of thromboembolism, more particularly the surgical conditions of thrombophlebitis and phlebothrombosis and of embolism, pulmonary and peripheral. In addition, attention is invited to the more accurate methods for

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From the Department of Surgery, Presbyterian Hospital of the City of Chicago, affiliated with the College of Medicine of the University of Illinois.

1. Barker, N. W.; Cromer, H. E.; Hurn, M., and Waugh, J. M.: Surgery **17**:207, 1945.

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5. (*a*) Olwin, J. H.: Unpublished data. (*b*) Owren, P. A.: Personal communication to the author.

6. Fahey, J. L.; Olwin, J. H., and Ware, A. G.: Proc. Soc. Exper. Biol. & Med. **69**:491, 1948. Olwin, J. H.: To be published. Owren.[5b]

controlling anticoagulant therapy, particularly the effects of dicumarol.[8] Time does not permit a comparison of the results of this type of therapy with the results of other methods.

## PRESENT STUDY

The present study is based on a series of 67 patients with peripheral phlebothrombosis or thrombophlebitis and 21 patients with embolism treated with dicumarol,[8] 47 of whom also received heparin initially. Of the emboli, 17 were pulmonary, 2 involved vessels in the extremities and 2 were cerebral. One of the patients with a cerebral embolus also had embolization of the vessels in the extremities.

TABLE 1.—*Anticoagulant Therapy in Phlebothrombosis and Thrombophlebitis*

| | Number of Cases | Pulmonary Emboli | | Fatal Pulmonary Emboli | | Absence of Residual Signs | | Patients Lost |
|---|---|---|---|---|---|---|---|---|
| | | No. | Per Cent | No. | Per Cent | No. | Per Cent | |
| Phlebothrombosis | 45 | 1 | 2.2 | 0 | 0 | 39 | 86.6 | 2 |
| Thrombophlebitis | 22 | 0 | 0.0 | 0 | 0 | 17 | 77.2 | 1 |
| Total | 67 | 1 | 1.5 | 0 | 0 | 56 | 83.5 | 3 |

TABLE 2.—*Anticoagulant Therapy in Cases of Embolism*

| | Number of Cases | Cases in Which Emboli Developed After Therapy Was Controlled | | Mortality |
|---|---|---|---|---|
| | | Number | Per Cent | |
| Pulmonary embolism | 17 | 1 | 5.8 | 0 |
| Peripheral embolism | 3 | 0 | 0.0 | 0 |
| Cerebral embolism | 2 | 0 | 0.0 | 0 |
| Total | 22 | 1 | 4.7 | 0 |

## HEPARIN AND DICUMAROL AND METHODS FOR CONTROLLING THEIR EFFECTS

Heparin is a rapidly acting drug and, according to present da[illegible] concepts, depends for its chief effect on the inhibition of the conversion of prothrombin to thrombin. The role of the prothrombin accelerator may be important in this process.[7]

Dicumarol[8] acts more slowly, requiring a minimum of forty-eight hours to achieve therapeutic effect, and its action is attributed to a depression of prothrombin formation. It would seem reasonable, there-

7. Owren, P. A.: Proc. Norwegian Acad. Sc., 1944.

fore, that efforts toward controlling the effects of both drugs might well be directed toward an accurate control of the active prothrombin level.

The common means for controlling heparin therapy has been a control of the coagulation time of whole blood. Because both the therapeutic and the safety ranges are rather broad, this has been reasonably satisfactory. The chief advantage of the test lies in its ease of performance. In different or even in the same hands, however, it is subject to unpredictable and unexplained variations, and its use is not without danger. Dosage of heparin in this study has been controlled by regulation of the measurable prothrombin level, and, as will be shown later, this factor is believed to be a more accurate reflection of heparin effect than is the coagulation time of the whole blood.

It is generally conceded that the only satisfactory control for dicumarol® effect is the estimation of the prothrombin level. Most hospitals rely on the one stage method of Quick [8] or modifications of it for the estimation of this factor. Because of the several uncontrolled factors other than prothrombin,[9] the test has led to errors in prothrombin estimation and may have been responsible for fatal hemorrhages from the use of dicumarol.® The two stage method described by Warner, Brinkhous and Smith [10] and recently modified by Seegers [11] has given more uniformly accurate results and has been the guide for dicumarol® therapy in this series of cases. With this method it has been possible to reduce the prothrombin to the generally approved range of 10 to 30 per cent and, with few exceptions, maintain that range over a period of months or years if desired.[5a] It is also possible with this method to carry on outpatient therapy with safety (fig. 1).

### ROUTINE OF THERAPY

When a diagnosis of thrombophlebitis or phlebothrombosis is made, the patient's prothrombin level is determined, and if it is in the neighborhood of 100 per cent, i. e., 85 to 105 per cent, he is given 300 to 400 mg. of dicumarol® by mouth, depending on the prothrombin level and his weight. The following day he is given 200 mg. At the time of the first dose 50 to 70 mg. of heparin is injected by vein, and fifteen to thirty minutes later another determination of prothrombin is made. A level of approximately 35 per cent is sought, and the dosage of heparin

8. Quick, A. J.: J. A. M. A. **110**:1658 (May 14) 1938.

9. Olwin, J. H.: Am. J. M. Sc., to be published.

10. Warner, E. D.; Brinkhous, K. M., and Smith, H. P.: Am. J. Physiol. **114**:667, 1936.

11. Seegers, W. H., in Trans. First Conference on Blood Clotting and Allied Problems, New York, Josiah Macy, Jr. Foundation, 1948.

is adjusted accordingly. The adjusted dosage is repeated every four hours until the dicumarol® effect is established.

The prothrombin level is determined on the third morning. It must be borne in mind that the effects of a single intravenous dose of heparin ordinarily persist for from four to six hours, and blood for estimation of the dicumarol® effect must be drawn at least six hours after the last intravenous dose of heparin (fig. 2). If the dicumarol® has not reduced the prothrombin to therapeutic levels at the end of forty-eight hours, supplemental injections of heparin in reduced amounts may be given until such effect is achieved. Subsequent doses of dicumarol® depend

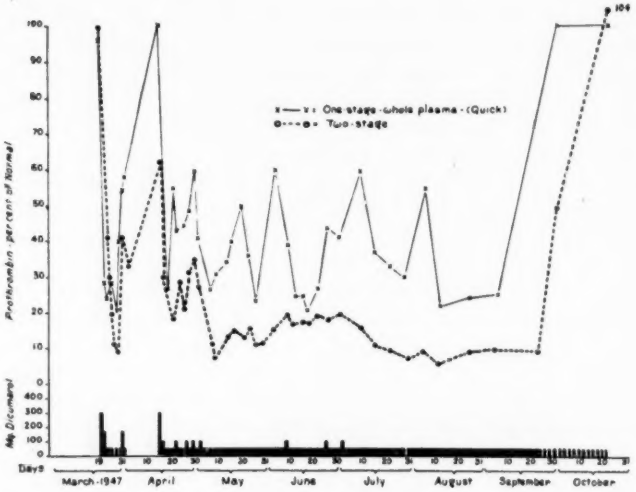


Fig. 1.—Comparative levels of prothrombin with one stage and two stage methods. This chart represents the course of therapy of a patient with thrombophlebitis of the leg which developed after minor trauma. When his prothrombin reached therapeutic levels, his condition improved and he elected to discontinue taking dicumarol. His signs returned when his prothrombin approached normal levels, and he was again placed on anticoagulant therapy. Because of continued slight swelling of the leg, he was maintained on dicumarol over a period of months, at the end of which time his leg appeared normal. He carried on normal office routine during the last six months of the therapy. Note the uniformity of the prothrombin estimations as shown by the two stage test and the wide variations in the one stage results. Had the latter been guiding the therapy, serious bleeding might have occurred and the prothrombin would not have been therapeutically controlled.

on the prevailing prothrombin level, and after the first four days of therapy determinations are usually made every other day and eventually once a week. In some patients who have been on therapy for over two years for other conditions estimations are made twice monthly.

Patients are kept in bed until the prothrombin is controlled, and in the case of phlebothrombosis they are then allowed up. If edema is present, an elastic stocking is provided. Those with the picture of thrombophlebitis are kept in bed until the more acute symptoms and signs subside and are then allowed to be up. The latter patients receive warm, moist dressings in the acute stages. Ligation of veins or sympathetic block has not been used. The use of dicumarol® is continued and the prothrombin maintained at therapeutic levels for one to three weeks after all signs have disappeared or until no further improvement is apparent. The minimum time of therapy has been two weeks, the maximum eight months and the average four to six weeks.

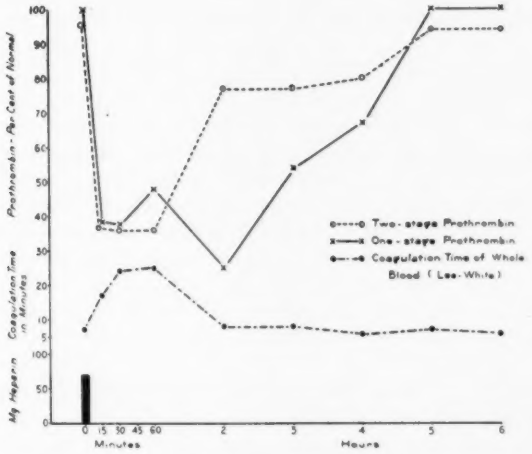


Fig. 2.—This chart represents the response of a patient to a single dose of heparin as shown by the prothrombin levels and the coagulation time of the whole blood. Note that the latter shows a return to normal after two hours, whereas the prothrombin remains somewhat depressed as long as four hours after the heparin injection. Prothrombin estimation is considered a more sensitive and uniformly accurate reflection of the effect of heparin than is the coagulation time of whole blood.

The program for cases with embolism is the same as that described with the exception that the prothrombin level is not obtained prior to the administration of heparin in acute cases. A large dose (70 to 100 mg.) of heparin is given through the same needle used for drawing blood and as rapidly as possible after the embolus occurs. Patients with embolism are allowed out of bed as soon as the prothrombin level is controlled, if other conditions permit.

## RESULTS

In the 45 patients with phlebothrombosis the usual duration of signs and symptoms was one to two weeks. In no patient did the full blown picture of thrombophlebitis develop. In 7 nonfatal pulmonary emboli had developed before administration of anticoagulants was started. Of the latter, 6 had no further embolic episodes. One patient suffered a nonfatal embolism after he was on controlled therapy. Another patient, in addition to the 7, had twice recovered from episodes of cerebral thrombosis and thirty-four days after discontinuation of the use of anticoagulants suffered a fatal pulmonary embolus at home from an unrecognized pelvic phlebothrombosis. Thirty-nine (86.6 per cent) of the 45 patients with phlebothrombosis had no residual signs after the passing of the acute episode. Two died of other causes and hence could not be followed, 2 are lost from observation and 1 had residual swelling.

Twenty-one patients had what was considered thrombophlebitis of the leg, arm or, in 1 instance, large veins of the abdominal wall. In 7 it involved only the superficial vessels. In 1 of them extension to the deep vessels developed after controlled anticoagulant therapy was established. A nonfatal pulmonary embolus developed in 1 patient with thrombophlebitis prior to the beginning of anticoagulant therapy and in none after it was started. Of the 22 patients with thrombophlebitis, 17 (77.2 per cent) had no residual signs after the acute phase subsided, 1 died of generalized carcinomatosis and hence could not be followed, 1 is lost and 3 have residual swelling. In general, the earlier administration of anticoagulants was started the shorter was the course of the disease (fig. 3).

Seventeen patients in this study were treated after the development of pulmonary emboli. The source of the emboli in 9 cases was phlebothrombosis and in 1 case thrombophlebitis, and in 6 cases they were considered cardiac in origin. (The patients in 4 of these cases had auricular fibrillation.) In 1 case the source of the embolus was undetermined.

One patient suffered one embolus after anticoagulant therapy was established. None of the emboli was fatal. One patient with both phlebothrombosis and anterior and posterior myocardial infarctions ceased having emboli when anticoagulant therapy was established. He died at home thirty-four days after the use of dicumarol® was discontinued from what appeared to be a cerebral embolus.

In 2 patients with auricular fibrillation cerebral emboli with hemiplegia developed, and of these, 1 also had embolization of the left axillary and the right brachial arteries. She was in coma for six days. Recovery has been progressive, however, and at the present time (six months after the use of anticoagulants was started) her speech and

the circulation and function of both arms have returned to normal. The patient walks with the aid of a cane, though the leg still shows about 30 per cent loss of function. The other patient has shown about 30 per cent improvement after three weeks of anticoagulant therapy.

Two patients with bacterial endocarditis and peripheral emboli had no further embolism while given anticoagulant therapy.

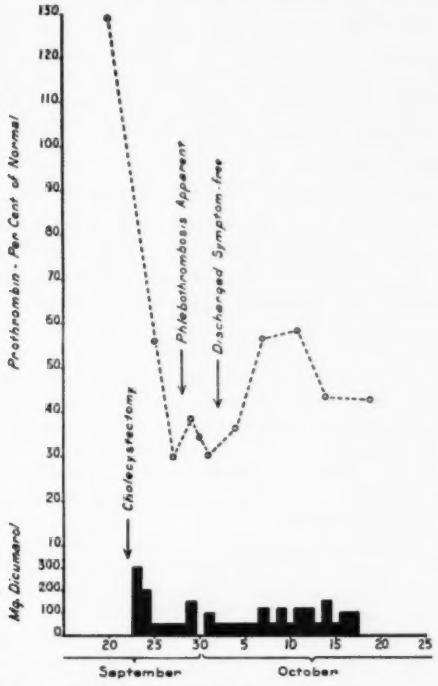


Fig. 3.—This chart shows the possible advantage of early institution of anticoagulant therapy. Because the patient represented had a prothrombin level of 129 per cent, even though there is no consistent correlation between an increased prothrombin level and intravascular clotting, administration of dicumarol was started on the second day after cholecystectomy. Four days later, at which time the prothrombin was considered controlled, swelling of the left leg and tenderness of the calf developed. The thrombus had no doubt been forming since the operation. All signs and symptoms had disappeared five days later, and the use of dicumarol was discontinued after fifteen additional days.

## BLEEDING HAZARD

The two stage determination being a highly accurate measure of prothrombin concentration, the bleeding hazard is minimized. In this series bleeding occurred in 7 cases (9.4 per cent), the most common

site being the urinary tract. Withdrawal of the drug will in many instances allow the prothrombin to rise to nonbleeding levels. Administration of vitamin K intravenously in doses of 24 mg. every two hours for three doses will usually result in a prompt rise in prothrombin. Moderate to severe hepatic damage, blood dyscrasias and pathologic changes in the kidney may be relative contraindications to the use of dicumarol,* and in such instances vitamin K may have little effect on the prothrombin level.

SUMMARY

Sixty-seven patients with phlebothrombosis or thrombophlebitis and 21 with embolism, a total of 74 patients, were treated with dicumarol,* and in 47 of the cases heparin was used initially. The early disappearance of signs and symptoms, the low incidence of complications and the absence of mortality recommend this type of therapy for further trial.

The accurate control of the effects of the two drugs is important. The methods in common use at the present time are considered inadequate both from the point of view of the maintenance of an effective therapeutic level of prothrombin and from the standpoint of safety.

## DISCUSSION

Dr. Don H. O'Donoghue, Oklahoma City: There are two things Dr. Olwin can help me with. In most of our cases thromboembolism occurs after rather massive orthopedic operations, such as disk and fusion or major operations on the hip. The other situation, in which we run into thrombophlebitis particularly, is in massive crushing injuries. The sites of both of these operations, as you can see, are places where it is difficult to have good hemostasis either postoperatively or post-traumatically.

In most instances the condition comes on seven to ten days, and even later, after surgical intervention. I should like to be informed as to whether this procedure of lowering the level to 10 to 35 is safe in these cases or whether one is likely to get serious hemorrhage after the administration.

Dr. John H. Olwin, Chicago: I think it depends entirely on the method of control. I believe our fatal accidents from hemorrhage have been a result of inadequate methods for controlling the coagulation factors.

The two stage method of Warner, Brinkhous and Smith, more recently modified by Seegers, is described completely in the "Transactions of the First Conference on Blood Clotting and Allied Problems," published by the Josiah Macy, Jr. Foundation, 565 Park Avenue, New York. It is more complicated than the one stage method, but it seems to me that the results are well worth the trouble required in setting it up.

Also, perhaps it is of interest to you that the reagents for this test are expected to be available commercially before long and will make the test considerably more simple to perform.

We have used this method of therapy within a few hours after a major surgical procedure, and in no instance have we had any serious hemorrhage. Using this method for control, we have had hemorrhage in about 10 per cent of all our

cases in which anticoagulant therapy was used, which now number about 200. This is usually from the urinary tract and may be microscopic or gross, lasting perhaps one to seven days depending on the therapy. Occasionally there is excessive bruising, some nasal bleeding, some bleeding from the gums and occasionally some hematomas intramuscularly.

Because we feel that the prothrombin level is always known and the patient is never far below his bleeding threshold, we have not been concerned about minor hemorrhage. Except in certain cases of hepatic or renal dysfunction or in certain blood dyscrasias, the prothrombin will return rather promptly to normal within a few hours after large doses of vitamin K are given. I would say that this should apply to orthopedic operations as well as to general ones.

# DIAGNOSIS AND SURGICAL TREATMENT OF PATENT DUCTUS ARTERIOSUS

WILLIS J. POTTS, M.D.
STANLEY GIBSON, M.D.
SIDNEY SMITH, M.D.
AND
WILLIAM L. RIKER, M.D.
CHICAGO

AT BIRTH the patent ductus arteriosus has fulfilled its function and should obliterate itself. Various reasons have been presented for its failure to close, but none have been convincing. Why so few ducti remain permanently open seems a more reasonable question. Why should a large endothelium-lined tube between vessels carrying different intraluminal pressures close? Until further evidence is gained all we can do is lamely ascribe the tendency toward spontaneous closure to the fundamental characteristic of the tissue.

Many ducti do not close promptly at birth. Christie [1] has pointed out that 44 per cent are still open at 1 month of age, 12 per cent at 2 months of age, 2 per cent at 8 months and approximately 1 per cent at 1 year of age. A few ducti close spontaneously between the first and second years of life. It is generally assumed that a ductus which is open at 2 years of age will remain permanently patent.

In a typical case uncomplicated by other cardiac abnormalities a diagnosis of patent ductus arteriosus can be made almost without error. The outstanding all-important physical finding is the classic "humming top" or "machinery" murmur. It is usually heard best at the second left interspace anteriorly. The murmur is continuous, going all the way through systole and diastole. In fact, if the murmur does not go all the way through diastole the patient very likely does not have a typical patent ductus arteriosus. Taussig said:[2] "The absence of a continuous murmur is strong contraindication for operation. With

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From the Children's Memorial Hospital.

Read at the Fifty-Sixth Annual Meeting of the Western Surgical Association, St. Louis, Dec. 2, 1948.

1. Christie, A.: Normal Closing Time of the Foramen Ovale and the Ductus Arteriosus, Am. J. Dis Child. **40**:323 (Aug.) 1930.

2. Taussig, H.: Congenital Malformations of the Heart, New York, The Commonwealth Fund, 1947, p. 349.

the possible exception of a subacute bacterial endocarditis, operation should never be considered in the absence of a continuous murmur over the base of the heart."

In this series of 52 cases the diagnosis was confirmed at operation in every case. Two patients had raucous systolic murmurs which had a slight carry-over into diastole. These patients were carefully studied

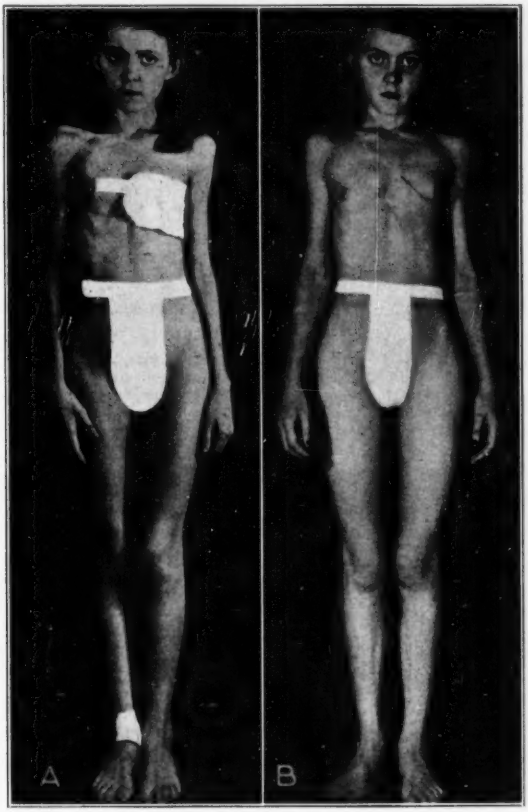


Fig. 1.—*A*, B. C., aged 13 years and weighing 65 pounds (29.5 Kg.), on the verge of heart failure, was operated on on Feb. 7, 1947. The ductus was huge, 18 mm. in diameter (outside measurements). *B*, the patient gained 22 pounds (10 Kg.) during the first two months following operation (see fig. 2).

by a number of cardiologists, and 1 patient was subjected to catheterization of the heart. Our conclusion in both cases was that an arteriovenous shunt existed but probably not a typical patent ductus. At operation

the findings in both were identical. There was no patent ductus but an abnormal opening in the common wall of the aorta and the pulmonary artery just beyond their emergence from the ventricles. It was a

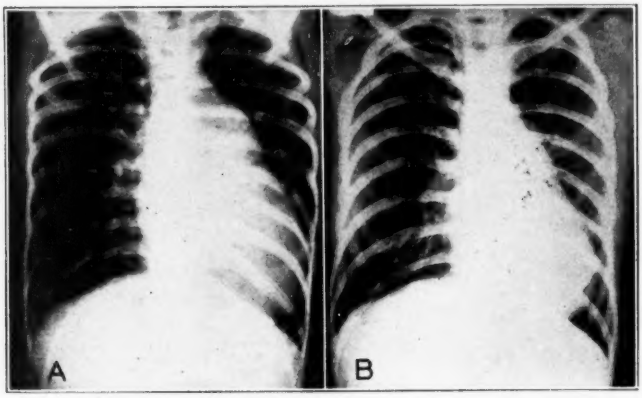


Fig. 2.—*A*, preoperative roentgenogram made on B. C. on Feb. 2, 1947, showing enlargement of the heart and marked dilatation of the pulmonary artery. *B*, roentgenogram taken on Nov. 13, 1948, twenty-one months after operation. In spite of normal physical growth and gain in weight the heart is smaller than before operation.

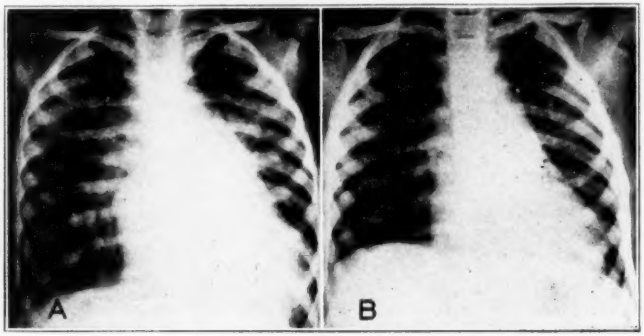


Fig. 3.—*A*, D. W., aged 4, weight 32 pounds (14.5 Kg.), height 41 inches (109 cm.), was operated on on Dec. 10, 1946. The outside diameter of the ductus was 12 mm. *A*, roentgenogram taken before operation shows cardiac enlargement and pronounced vascularity of the lung fields. *B*, this roentgenogram taken twenty-two months after operation shows that the heart is smaller than before operation. The vascularity of the lung fields had disappeared six weeks after operation.

temptation to attempt a suture closure of this opening. The hazard of such a hitherto untried procedure and the probability of the closure,

at best imperfect, being inadequate and tending to reopen made us regretfully abandon the idea.

Palpation of the chest over the left pulmonic area will often reveal a thrill which one child unprompted described as follows: "It feels like bees in a bag." The simile is excellent. The intensity of the thrill is roughly indicative of the size of the ductus.

The systolic blood pressure is usually normal, but the diastolic pressure is apt to be low—often in the vicinity of 30 to 50 mm. of mercury. Consequently there is a water hammer type of pulse, and a "pistol shot" can be felt over the femoral arteries. A capillary pulse can be seen in the lower lip pressed down firmly with a glass slide. This sign is far from infallible, because a similar pulse can often be seen in children whose hearts are normal.

If the patent ductus is large, physical development is frequently retarded (fig. 1). The height, but especially the weight, is apt to be subnormal. However, many of the children seem to be especially alert mentally and, so long as they are not impeded by heart strain, are hyperactive. This desire for constant activity has been observed often enough to suggest that it may have a cause.

A child with an uncomplicated patent ductus arteriosus is never blue.

In 3 of 52 patients there have been associated congenital cataracts. The mother in each instance had had rubella during the early months of pregnancy. This finding is significant in view of the attention that has been focused on rubella in the early months of pregnancy as a cause of certain congenital abnormalities.

Of the routine laboratory studies, the roentgenogram alone gives valuable information. The red blood cell count is normal. The electrocardiogram is regularly normal or may show a slight deviation of the axis to the left or right. If the ductus is small, the heart shadow is usually normal or nearly so. In patients who have a large ductus the heart is enlarged (fig. 2) and a bulge is seen at the region of the left base. This enlargement is due to dilatation of the pulmonary artery. Vascularity of the lung fields (fig. 3) due to excessive blood flow in the lesser circulation is seen in the roentgenogram, and a "hilar dance" can be seen fluoroscopically.

## SURGICAL CONSIDERATIONS

Indications for operative closure of the patent ductus have slowly broadened as the mortality of surgical therapy has lessened. Most cardiologists today advise that all typical patent ducti be treated surgically. Even though the child has no symptoms and the condition is discovered incidentally, operation should be advised because of the risk

of eventual irremediable complications is greater than that of surgical intervention. It is not uncommon for a patient of middle age with a known patent ductus urgently to request operation because of rather sudden appearance of signs of heart failure. At this age the operative mortality is high and the operation is difficult, whereas in children the mortality is low and the operation when mastered is not too difficult. With each decade the ductus becomes shorter, adhesions about it more dense, the walls more rigid and friable and consequently the danger of uncontrollable hemorrhage greater. Operation is therefore advised for all typical patent ducti in children between the ages of 2 and 12 years.

Cardiologists advise surgical treatment, but surgeons are not in agreement as to the best method of obliterating the ductus. Originally Gross [3] employed simple ligation, but as experience increased and recurrences presented themselves he advised division and suture. At present he severs all patent ducti.[4] Blalock,[5] on the contrary, stated the belief that ligation is the treatment of choice. Shapiro and Johnson [6] studied 643 patients who underwent operation for patent ductus by 46 surgeons. In 525 the ductus was uninfected, in 101 it was infected and in 17 the diagnosis was erroneous. The mortality rate in cases of noninfection was 4.9 per cent, and the rate of recurrence was 8.7 per cent. They concluded: "Ligation of the ductus is now obsolete and should be done only where section is impossible. That section of the ductus is a safe and practical procedure is established by the fact that 172 patients with uninfected patent ductus arteriosus have been so treated without a single fatality and no recanalization."

Gilchrist [7] reported on a boy of 13 with a large ductus, 1 cm. long and 1 cm. broad, ligated with two double silk ligatures. A systolic murmur was detected three days after operation, and at seventeen days all local signs of a patent ductus arteriosus were again fully developed. He also cited moderate improvement in 2 cases in which the ductus was incompletely occluded by ligature.

From a survey of medical literature and from discussions by men of experience in the field of surgery of the ductus it appears that

3. Gross, R. E., and Hubbard, J. P.: Surgical Ligation of Patent Ductus Arteriosus: Report of First Successful Case, J. A. M. A. **112**:729 (Feb. 25) 1939.

4. Gross, R. E.: Complete Division of Patent Ductus Arteriosus, J. Thoracic Surg. **16**:314 (Aug.) 1947.

5. Blalock, A.: Operative Closure of the Patent Ductus Arteriosus, Surg., Gynec. & Obst. **82**:113 (Feb.) 1946.

6. Shapiro, M. J., and Johnson, E.: Results of Surgery in Patent Ductus Arteriosus, Am. Heart J. **33**:725 (May) 1947.

7. Gilchrist, R. R.: Ligation of Ductus Arteriosus, Edinburgh M. J. **53**:346 (July) 1946.

reconsideration of the question of ligation versus division is in order. A number of facts must, therefore, be considered: Incomplete occlusion of a ductus by an insufficiently snug ligature must be regarded as an operative failure; too tight ligation may result in hemorrhage at the time of operation or in recanalization later; it is generally agreed that infected ducti should be divided; the best principles of surgery are not employed when one buries large amounts of foreign material against large vessels and bronchi in children or adults; ligation appears to be an easier and safer procedure in the hands of most men; whether operative mortality is greater after division than after ligation is not definitely known; if the mortality is lower after ligation, does that justify poorer final results while waiting for time and experience to decide whether ligation or division of a patent ductus is the operative procedure of choice? At present, if compromise is permissible, this might be considered. A surgeon who has had adequate training and experience and who has familiarized himself with the handling of large vessels probably should employ the technic of proper ligation in his early cases. Surgery of the ductus is not easy, and even a surgeon with the stoutest nerves and the steadiest hands will fumble a bit in his first few cases. After he has developed confidence in his ability to dissect the ductus completely free and has convinced himself that he can properly suture a large vessel, then the hurdle of division and suture will not seem too high. Furthermore, it is well for the beginning surgeon to remember that surgery of the ductus is far less formidable in children than in adults.

Without question the only obstacle to universal acceptance of division and suture of a ductus is the well founded danger of uncontrollable hemorrhage due to slipping clamps. It appears that a recently developed ductus clamp employing a new principle may greatly lessen this danger.[8] This clamp (fig. 4) has been used satisfactorily in the division of ducti in 30 consecutive cases. The principle of this clamp or forceps is embodied in rows of extremely fine teeth in apposing jaws. The teeth are approximately 1 mm. long and closely spaced—40 to 1 inch (2.54 cm.). The jaws of the clamp are about 1 mm. wide and hollow ground, so that they are less than 1 mm. wide at the base of the teeth. The hub of the clamp is so built that when the jaws are closed the teeth appose but do not interdigitate. The tiny teeth embed themselves in the adventitia of the vessel. In the experimental laboratories at Northwestern University these clamps were tested on large and small arteries and veins. It was demonstrated that they will occlude,

8. Potts, W. J.: A New Clamp for Surgical Division of the Patent Ductus Arteriosus, Quart. Bull., Northwestern Univ. M. School **22**:321 (Nov.) 1948.

will not slip and will not pierce or otherwise injure a vessel. Modified sizes and shapes of this clamp have been used satisfactorily for other types of surgery such as coarctation of the aorta, embolectomy and shunting operations for the relief of pulmonary stenosis.

## SURGICAL TECHNIC

Although the technic of division and suture of a patent ductus has been described previously, a few details are worthy of emphasis (fig. 5).

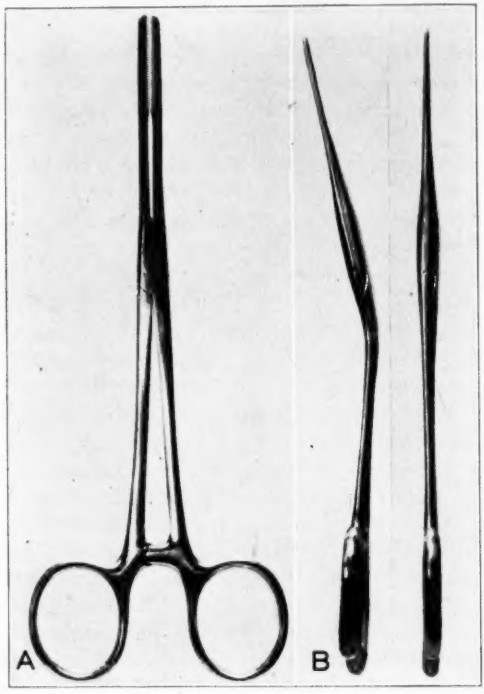


Fig. 4.—*A*, ductus clamp showing tiny teeth in apposing jaws. *B*, the angled clamp is placed on the aortic end of the ductus and the straight clamp on the pulmonary end.

We prefer a posterolateral incision through the left fourth interspace to the anterior submammary approach.

After the mediastinal pleura is opened, the fat, loose tissue and lymph glands lying on and about the ductus should be thoroughly removed. Constantly clear visualization of the field is an absolute essential. The vagus nerve and its recurrent laryngeal branch are

identified and retracted gently laterally. Cutting a few small obstructing branches of the vagus to the root of the lung seems to cause no ill effects.

The most important step in the operation is complete, thorough and painstaking dissection of the ductus. The lappet of pericardium overlying part or all of the ductus is grasped with a plain tissue forceps and with a scissors is dissected free. This dissection will be greatly simplified if it is made through the loose adventitial layers. It will be known that the dissection is being made through this layer if the

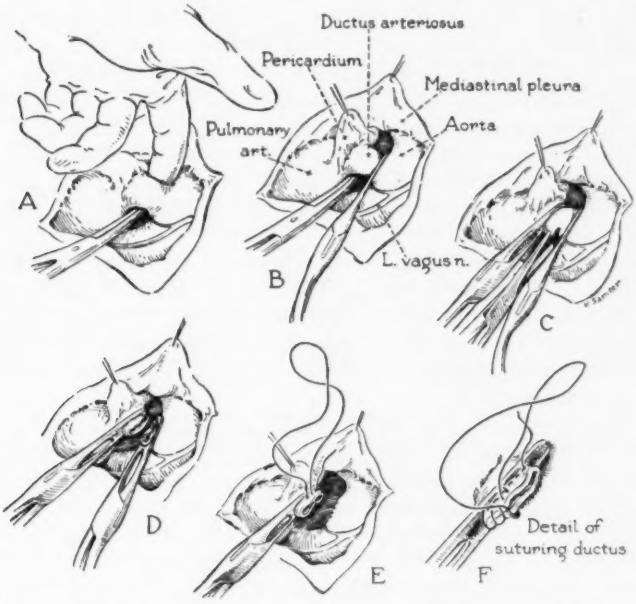


Fig. 5.—Technic of division and suture of patent ductus arteriosus.

gray-white wall of the ductus becomes visible. It cannot be too strongly emphasized that the lappet of pericardium overlying the ductus and the fibrous tissue about the ductus must be dissected completely free. After the ductus has been elevated from the underlying bronchus it is held up with a strip of umbilical tape so that the loose and fibrous tissue can be freed from its inferior surface also.

It will be discussed later under complications but the emphasis of repetition is justified—it is absolutely essential that the ductus be dissected free from all overlying loose tissue or any clamp will slip.

If a ductus is cleaned the teeth of the clamp will hold safely and surely in the wall. However, if the clamp is placed on the ductus in overlying loose tissue or if the pericardial fold is included in the clamp the cut ends of the ductus will tend to retract out of the jaws of the clamp. This danger is still greater when ordinary hemostats are used.

It is surprising how many ducti which at the beginning of dissection appear to be direct communications between the aorta and the pulmonary artery have a fairly reasonable length when dissection is complete.

The ductus is slightly elevated with a cystic duct forceps, and the ductus clamps are applied well on the aortic and pulmonary ends. These clamps must not be slid over the ductus or their edges will act as a saw. With the jaws wide open, the site of application is visualized and the clamp closed. Since the clamps are very thin, practically the entire length of the ductus, from 4 to 7 mm., lies between them. The ductus is cut and the stumps sutured with 5-0 Deknatel silk as illustrated in figure 5. The to-and-fro mattress suture followed by an over-and-over suture gives a nearer leakproof closure than a double row of continuous sutures.

After the aortic stump has been sutured, the clamp is partially opened to see if there will be any bleeding. The clamp is again closed in position and an extra stitch put in if necessary. The clamp is used rather than a retractor to hold the aorta out of the way while the pulmonary end is being sewed. Traction on the suture line in the aorta is thereby avoided.

Before closure we routinely place a snugly fitting catheter drain through the sixth interspace. This drain is connected with a water seal bottle after the patient is returned to bed and left in place two to three days. The advisability of using drains in such cases has been questioned. We have not had to aspirate the chest postoperatively and have had no infection in the chest or about the tube.

Postoperative care is routine. Penicillin in 100,000 unit doses is given twice a day for five to seven days. The patient is discharged from the hospital on the eighth to tenth postoperative day.

### OPERATIVE AND POSTOPERATIVE COMPLICATIONS

Hemorrhage from the ductus during operation occurred four times in 52 operations. In the second patient operated on a hole was poked in the anterior wall of the ductus with a cystic duct forceps while the dissection was being done. We should have used scissors and followed the proper line of cleavage instead of trying to do blunt dissection. Hemorrhage followed too tight ligation in the fourth patient operated on.

In case 38 the pericardium was not sufficiently well dissected from the pulmonary end of the ductus. The clamps were applied and the ductus cut. While the aortic stump was being sutured the inferior edge of the pulmonary end of the ductus slowly rolled out of the lower end of the clamp. It was easily caught with a stitch, but the need of adequate clearing of the ductus before applying the clamps was a lesson vigorously taught. Hemorrhage in case 50 was caused by opening a small aneurysm on the posteroinferior border of the ductus, where visualization was poor. Clamps were placed on each side of the small opening and the ductus was cut and sutured satisfactorily.

Although the hemorrhage was severe in case 2, slight in the other 3 and controlled in all without much difficulty, in each instance there were anxious moments. Hemorrhage in the first 3 instances was due to faulty technic and in the fourth to an unexpected, previously undemonstrable, pathologic process.

The recurrent laryngeal nerve was injured in cases 2 and 4 through hasty and panicky application of hemostats to a bleeding ductus.

Postoperative complications have not been serious. Three children had postoperative atelectasis of the left lung; 2 recovered spontaneously, and the third recovered promptly after bronchoscopic aspiration. One child had pneumonia of the lower lobe of the left lung. Two children had severe tracheobronchitis, probably due to the irritation of intratracheal anesthesia. One of these children required tracheotomy. Urticaria in 1 patient was presumably due to penicillin sensitivity.

## DISCUSSION

The size of the patent ductus, with one exception, varied from 4 to 12 mm. in diameter, with an average of 7.4 mm. These are outside measurements made with a caliper. Many ducti had the shape of a truncated cone, wide at the aortic end and tapering toward the pulmonary end. One patient (fig. 1) had a ductus actually measuring 18 mm. in diameter. This is the largest ductus of which record can be found. She was 13 years old and weighed 65 pounds (29.5 Kg.). Her heart (fig. 2) was enlarged and showed an unusually large convexity at the left base. Her chest heaved with each heart beat. She was on the verge of cardiac failure. She gained 22 pounds (10 Kg.) in weight during the first two months following operation.

As would be expected, after closure of a ductus in a child with cardiac enlargement the heart retains its size until body growth catches up with it. In a few patients who had considerable enlargement of the heart at the time of operation and who were, in fact, at the brink of heart failure, the size of the heart is smaller now than it was two years ago, before operation.

Mortality is a perilous subject to discuss. A well known surgeon lamented his expressed pride in 150 consecutive operations on the gallbladder performed without a fatality. The next 2 patients died. The mortality rate for surgery of the ductus, especially in children, is and should remain low. In this series of 52 cases there has been no death. All the patients were between 2 and 13 years of age.

## SUMMARY

Diagnostic features of patent ductus arteriosus are presented. Importance of the characteristic continuous murmur is emphasized. The subject of ligation or division and suture of the ductus is discussed. It is believed that by use of a recently described ductus clamp embodying a new principle the danger of uncontrollable hemorrhage can be lessened. In this series of 50 consecutive cases, 20 ducti were ligated and 30 ducti were divided and sutured. There were no deaths.

# REPAIR OF DEFECTS IN ETHMOID AND FRONTAL SINUSES RESULTING IN CEREBROSPINAL RHINORRHEA

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THE PURPOSE of this contribution is to call attention to the etiologic factors responsible for the spontaneous development of cerebrospinal rhinorrhea and the methods employed in closing defects in the cribriform plate of the ethmoid bone which were congenital in origin or the result of bullet wounds and to review the results of our surgical experiences in treating cerebrospinal rhinorrhea.

## CAUSATION

Cerebrospinal rhinorrhea may result from a number of causes, the most common of which is skull fracture that extends through the posterior wall of the frontal sinus (fig. 1) or the cribriform plate of the ethmoid bone, with accompanying tears of the dura and arachnoid.[1] The first evidence of rhinorrhea associated with fracture of the skull is the occurrence of a watery, bloody discharge from the nose. In most instances, in our experience, the lesions heal, with spontaneous remission of the rhinorrhea. Persistent rhinorrhea or its delayed occurrence usually is due to considerable loss of bone, absorption of a fragment of bone or inclusion of the dura and arachnoid between fragments of bone permitting escape of cerebrospinal fluid into one of the nasal cavities. Cerebrospinal rhinorrhea frequently has occurred spontaneously as the result of a congenital defect in the cribriform plate which permits extension of an envelope of dura and arachnoid along an olfactory nerve fiber through the cribriform plate. In such cases leakage of cerebrospinal fluid undoubtedly is the result of rupture of the thinned-out arachnoid and mucous membrane. According to reviews in the literature, precipitation of this type of rhinorrhea usually is the result of coughing or sneezing during an attack of head cold. Rhinorrhea has accompanied hydrocephalus, and in the particular instance

From the Section on Neurologic Surgery, Mayo Clinic.

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1. Brody, B. S.: J. Mt. Sinai Hosp. **5**:444, 1938. Gupta, N.: Indian M. Gaz. **71**:264, 1936.

leakage of cerebrospinal fluid was the result of increased intracranial pressure on thinned-out membranes and absorptive defects of the cribriform plate. We have also seen the condition afflict 2 patients suffering from pituitary tumor. The floor of the sella turcica had been absorbed, with presumable leakage of cerebrospinal fluid into the sphenoidal sinus. We also observed the condition in a patient who had a large osteoma of the orbit which had extended through the ethmoid into the anterior fossa. Rhinorrhea has followed the removal of nasal polyps. The polyps were meningoceles that had extended through the cribriform plate. Rhinorrhea also has resulted from craniotomy when, in the making of the anterior margin of the bone flap, the frontal sinus was unintentionally opened.

### SYMPTOMS AND SIGNS

The only symptom that many patients complain of is an annoying, watery discharge of the nose. It may be continuous, or it may cease for

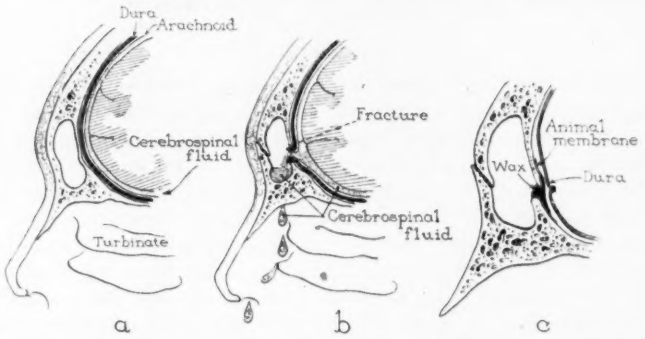


Fig. 1.—*a*, normal relationships of the brain, meninges and skull in the frontal region; *b*, craniosinusal fistula, with leakage of cerebrospinal fluid into the frontal sinus and thence into the nose; *c*, plastic closure of the meninges and occlusion of the bony defect with wax.

only a few hours and then resume. The discharge may appear as drops of clear fluid, or it may pour out in a stream when the head is tilted in certain positions. Usually, it appears in one side of the nose and produces the sensation that is experienced when the nose is congested. In cases of acute injury of the head, it may be difficult to recognize cerebrospinal fluid because it is mixed with blood, but when the condition is chronic the fluid is watery and can be readily collected and examined chemically. Cerebrospinal fluid is colorless, limpid and slightly viscous, has a specific gravity of 1.004 to 1.008 and contains traces of protein and small quantities of inorganic salt and dextrose. The lymphocyte count varies from 5 to 10 cells per cubic millimeter of

fluid. If there is doubt about the presence of cerebrospinal fluid in a discharge from the nose, the indigo carmine test, in which 1 cc. of indigo carmine is introduced into the subarachnoid space of the spinal canal by means of routine spinal or cisternal puncture, might be employed. Since the dye promptly stains the cerebrospinal fluid a bluish green color, it will be seen to appear in the nasal discharge within about fifteen minutes and will continue for two or three hours if fluid discharging from the nose contains cerebrospinal fluid. This test is of particular value in the differentiation between cerebrospinal rhinorrhea and allergic or vascular rhinorrhea. Roentgenologic examination of the skull offers some aid in the diagnosis in cases of recent fracture, but rarely is such examination of much assistance in the localization of cerebrospinal fistula involving the ethmoid cells.

Meningitis is the serious complication which may accompany this condition. It occurs more often when rhinorrhea results from fracture of the skull than it does when rhinorrhea occurs spontaneously, but it is a constant threat in all cases, and especially so when the patient has contracted a nasopharyngeal infection. Fortunately, chemotherapy has proved of value in combating meningitis and also has made it possible to perform these extensive operations without fear of the development of this complication.

### CONSERVATIVE TREATMENT

Conservative treatment (nonoperative) often has been employed by those who feared to advise or employ the radical procedure designed to close the fistulous tract. The argument set forth by those who advocate conservative treatment is that they believe it safer not to disturb the patient than it would be to hazard a radical operation. Unfortunately, there is little to offer in the way of nonoperative treatment. Fox,[2] and Friedberg and Galloway [3] reported 2 cases in which spontaneous cerebrospinal rhinorrhea was controlled by the application of a 20 per cent solution of silver nitrate to the polypous area of the nose. Fox identified the opening through the cribriform plate by the use of indigo carmine, after which he applied the solution of silver nitrate several times, with apparent recovery of the patient. Friedberg and Galloway stated that they observed the cerebrospinal fluid entering the nasal cavity at a point at which the anterior third portion of the middle turbinate bone joins the middle third portion of this bone, at which point they applied a 20 per cent solution of silver nitrate. The discharge ceased within twelve days, and at the time of the report the patient had been free of rhinorrhea for fourteen months.

2. Fox, N.: Cure in Case of Cerebrospinal Rhinorrhea, Arch. Otolaryng. **17**:85 (Jan.) 1933.

3. Friedberg, S. A., and Galloway, T. C.: Ann. Otol., Rhin. & Laryng. **47**:792, 1938.

Conservative treatment is of definite value in cases of acute rhinorrhea accompanying fracture of the skull, since a number of the meningeal defects will heal spontaneously. Teachenor,[4] Dandy,[5] Cairns,[6] Munro[7] and Coleman[8] have advocated surgical repair of the meninges if rhinorrhea does not disappear within four to six days after the time of injury. The approach employed is a transfrontal sinus approach in which the dura is exposed at the point of injury. Teachenor has suggested removal of the posterior wall of the frontal sinus, along with craniotomy. Lesions involving the ethmoid bone and the overlying meninges have been dealt with more conservatively than this in cases in which the condition is acute. However, Coleman[8] has employed unilateral frontal craniotomy in repair of recently injured meninges over the ethmoid bone. It has been our experience that if spontaneous recovery from cerebrospinal rhinorrhea is to take place after fracture of the skull it may not do so for an interval extending from a few days to eight weeks, and, therefore, it has been our practice to limit the intake of fluid to 1,500 cc. per day, in order to decrease the output of cerebrospinal fluid, and to administer penicillin and sulfanilamide or sulfathiazole in doses of 60 to 90 grains (4 to 6 Gm.) per day until the concentration of sulfanilamide in the blood registers from 8 to 12 mg. per hundred cubic centimeters. Also, we have found it advantageous to have the patient remain in bed in a semierect posture, making sure that he sleeps in the same position, since this minimizes the flow of cerebrospinal fluid into the nose. In the event that conservative measures fail to effect spontaneous remission of the rhinorrhea, we should advise the procedure developed by one of us (Adson[9]).

### SURGICAL TREATMENT

Although fractures of the skull are common, in only a relatively small group of cases does cerebrospinal rhinorrhea develop. In 940 cases of trauma of the head which Coleman[8] reviewed, "there were 216 fractures of the skull, with eighty-seven fractures of the base. Of the eighty-seven basal fractures, fifteen involved the frontal sinuses; six of these were associated with severe compound, depressed fracture of the frontal vault and operation was promptly done for disinfection,

4. Teachenor, F. R.: Intracranial Complications of Fracture of Skull Involving Frontal Sinus, J. A. M. A. **88**:987 (March 26) 1927.

5. Dandy, W. E.: Pneumocephalus, Arch. Surg. **12**:949 (May) 1926.

6. Cairns, H.: J. Laryng. & Otol. **52**:589, 1937.

7. Munro, D.: New England J. Med. **213**:893, 1935.

8. Coleman, C. C.: Fracture of Skull Involving Paranasal Sinuses and Mastoids, J. A. M. A. **109**:1613 (Nov. 13) 1937.

9. Adson, A. W.: Proc. Staff Meet., Mayo Clin. **16**:385, 1941. Ann. Surg. **114**:697, 1941.

débridement and closure of the dura." Coleman's observations are similar to those of others. The incidence of rhinorrhea after fracture of the skull varies from 2 to 5 per cent. In view of the fact that rhinorrhea sometimes is spontaneously cured, we are inclined to wait six or eight weeks before advocating repair of the craniosinusal fistula unless the fracture is compound and involves both walls of the frontal sinus.

The previously accepted procedures for closure of craniosinusal fistulas which communicate with the frontal sinus, the ethmoid cells and the nasal cavity have been (1) in cases in which the condition is acute, surgical repair through the frontal sinus; (2) in cases in which the condition is chronic and it is possible to identify the site of the lesion, performance of small unilateral transfrontal craniotomy, identification of the fistula and closure of the meningeal opening with interrupted silk sutures or closure of it with sutures and covering of it with muscle, and (3) performance of unilateral transfrontal craniotomy, in which the dura is elevated, the opening is identified and a wick of iodoform gauze is placed between the lacerated dura and the cribriform plate, as Peet[10] has advocated. The end of the gauze wick is brought out through the frontal incision, but the wick itself is left in place for four days and then carefully removed. Peet stated: "The object of this procedure is to prevent meningitis by allowing the brain to become firmly adherent to the lacerated dura thereby effectively closing off the subarachnoid space before organisms passing through from the nose can cause infection."

One of us (Adson) has used these accepted procedures with varied success; the one difficulty that has been encountered in attempting to free the dura from the cribriform plate is that the dura has had a tendency to tear, as Grant emphasized in his discussion of Coleman's paper. The dura, in addition to being thin, is likewise under moderate tension. This prevents the carrying out of proper overlapping of the dura, which is necessary for thorough invagination of the meningeal fistulous tract. We have included muscle in the suture line to assure against leakage of cerebrospinal fluid. This failure prompted one of us (Adson) to develop the operation employed in these cases of chronic rhinorrhea.

The procedure consists of performance of craniotomy which will allow the dura to be elevated from the bone in both halves of the frontal fossa. The bone flap must be designed so as to extend across the midline and to uncover the anterior poles of both frontal lobes (fig. 2*a*). A coronal scalp flap incision is employed. It is placed within the hairline, after which the scalp and periosteum are reflected

10. Peet, M. M.: New York State J. Med. **28**:555, 1928.

forward to a line just above the frontal sinus. Six trephine openings are made, the first two of which are placed on each side of the midline just above the frontal sinus. The second two trephine openings are placed on each side of the midline and the longitudinal sinus, approximately 3 cm. in front of the coronal suture. The third two openings are placed in the temporofrontal region, one on each side. As the bone between all the openings is cut with a Gigli saw, an opening is effected which is sufficient to permit elevation of the dura and the frontal lobes. The bone flap is kept sterile during the operation by means of a

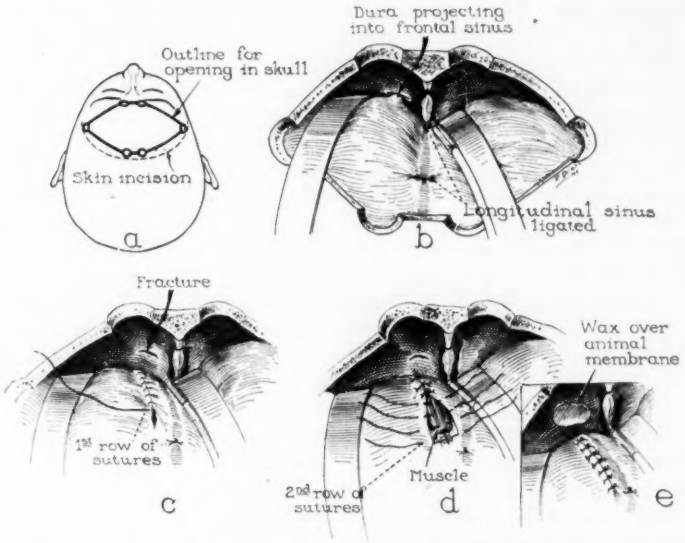


Fig. 2.—*a*, schematic outline of incision in the scalp and craniotomy; *b*, elevation of dura, identification of the fistulous tract and ligation of the longitudinal sinus; *c*, closure of the dural tear with a suture of continuous surgical gut; *d*, reenforcement of the primary closure by means of a second row of interrupted silk sutures, including a strip of muscle employed as additional protection against recurrence of the rhinorrhea; *e*, closure of bony opening with animal membrane and bone wax.

sponge soaked with isotonic sodium chloride solution. Caution is taken to avoid injury to the longitudinal sinus. Bleeding from the longitudinal sinus is controlled by ligation of the sinus (fig. 2 *b*) with silk ligatures at a level 6 cm. above the cecal foramen, which is situated superior to the crista galli. In some instances it is necessary to ligate the longitudinal sinus where it communicates with the cecal foramen. The advantage of ligation of the longitudinal sinus is that it allows the dura to be

sutured into and, if necessary, to be used in closure of, the fistulous tract. The procedure is continued by elevation of the dura from the frontal fossa and the olfactory grooves, where it is necessary to sacrifice the olfactory nerves. The elevation of the dura is continued until the anterior crest of the sella turcica is approached. During this dissection, the fistulous tract is always encountered, whether it be situated on the right or the left side. The meninges will be seen to extend into the defect of the frontal sinus or the cribriform plate.

After the dura has been mobilized sufficiently and the fistulous tract has been identified, plastic closure of the tract is begun by overlapping the dura in such a way as to invaginate the meningeal portion of the fistula. The first suture is placed in the most dependent part of the elevated dura (fig. 2*c*). This suture is continuous chromic surgical gut no. 0. The primary line of suture is protected by a strip of muscle which is transfixed to the dura and further reenforced by the placing of a second row of interrupted silk sutures (fig. 2*d*). The defect in the frontal sinus or cribriform plate is filled with Horsley's bone wax. To protect further against forcing of the wax through the cranial defect into the sinus or nose, Lukens' animal membrane is placed over the defect before the introduction of wax to plug the hole (fig. 2*e*).

The advantages of bifrontal craniotomy are as follows: 1. A better exposure is obtained than by employment of a unifrontal flap. 2. The surgeon is always sure to identify the fistulous tract. 3. The exposure thus obtained affords a better opportunity for elevation of the meninges along the cribriform plate. 4. After elevation of the dura from the cribriform plate, tension on the dura is relieved; this permits greater ease of invagination of the fistulous tract and successful performance of overlapping dural closure of meningeal defects.

In addition to the cases that we have encountered in which spontaneous rhinorrhea developed because of large openings in the cribriform plate with the resulting protrusion of meninges forming a nasal meningocele which has not been recognized by the otolaryngologic surgeon, we also have observed a number of cases in which cerebrospinal rhinorrhea has developed as the result of the removal of a nasal polyp which in reality was a nasal meningocele (figs. 3 and 4). In 1 of our cases rhinorrhea developed after the removal of a large meningioma of the olfactory groove which had eroded the cribriform plate.

Another patient had been wounded with a .38 caliber revolver. The bullet entered the right temporal area, followed the wing of the sphenoid, perforated the cribriform plate and passed on through the left superior maxilla. The opening in the cribriform plate was larger than 1 cm. in diameter, with resulting profuse cerebrospinal rhinorrhea (fig. 5*a*). This situation prompted one of us (Adson) to modify his previous

procedure by introducing a plate of tantalum. The usual bilateral transfrontal craniotomy was employed. Loose fragments about the defect were removed. The tip of the crista galli was resected, after which the defect was covered with a layer of Horsley's bone wax. On top of this first layer of wax a layer of tantalum was cut and molded to bridge across the cribriform plate. The plate was intentionally perforated in order to encourage the wax to come through these openings to lock it in place and prevent it from slipping (fig. 5 *b* and *c*). A second

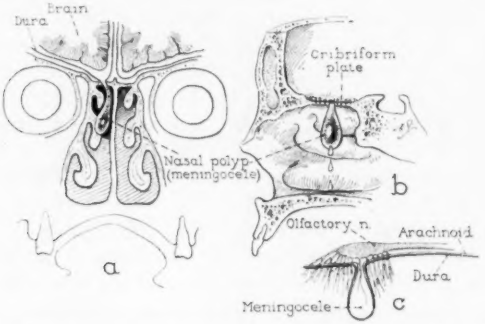


Fig. 3.—*a* and *b*, the course of a nasal meningocele; *c*, the relationship of the meningocele to the olfactory nerve.

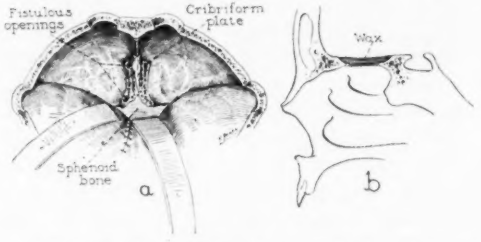


Fig. 4.—*a*, multiple fistulous openings in the cribriform plate; *b*, the use of Horsley's bone wax to obliterate the fistulous openings.

layer of wax was then placed over the top of the plate, molding it rather firmly into what had once been the olfactory grooves (fig. 5 *d*). In order to fix this plate further, it was heated by the use of the electrocoagulating current and needle. As the heat was applied, one could see the wax melt and subsequently mold into place.

Since the introduction of the tantalum plate along with the wax, which was first performed on Sept. 27, 1945, the method has been employed by Dr. Craig in 3 additional cases when he was dealing with

cribriform defects in the ethmoid bone. He has also used polythene as a covering for the fistulous opening in the meninges. Dr. Love and Dr. Baker on 2 occasions have used fibrin foam in place of Horsley's bone wax. Gelfoam® has also been used in addition to the wax for the purpose of hemostasis and for the stimulation of granulation about the

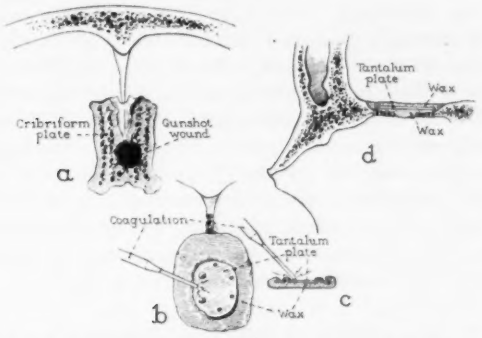


Fig. 5.—*a*, bullet wound in the cribriform plate; *b*, and *c*, insertion of the tantalum plate and the application of the electrocoagulating current in order to heat the plate and melt the wax to prevent slipping of the tantalum plate; *d*, relative positions of tantalum plate and bone wax.

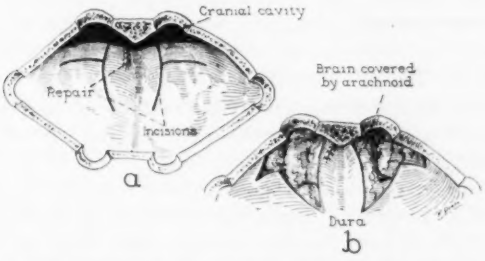


Fig. 6.—*a* and *b*, incisions in the dura employed in order to permit the arachnoid-covered frontal lobes to fill the anterior fossa.

fistulous opening. Since a space may remain between the dura and the anterior wall to the frontal bone when the dura is plicated to close the fistulous opening, one of us (Adson) has made elliptic incisions in the dura parallel and lateral to the longitudinal sinus and has added to them a lateral incision under each frontal lobe in order to allow the frontal lobes with arachnoid covering to expand forward and obliterate this space (fig. 6).

The accompanying table illustrates the incidence of cerebrospinal rhinorrhea in 26 cases and emphasizes that it occurs spontaneously twice

as frequently in females as it does in males and that in all cases the spontaneously developed rhinorrhea resulted from defects in the cribriform plate.

### A REVIEW OF CASES IN WHICH OPERATION WAS PERFORMED

Traumatic cerebrospinal rhinorrhea occurred in 8 cases in which operation was performed. In 6 of these the rhinorrhea was the result of fractures extending through either the frontal or the ethmoid bone. In 1 case cerebrospinal rhinorrhea developed as the result of a large meningioma of the olfactory groove, and in 1 case it developed as the result of a bullet wound. Cerebrospinal rhinorrhea had been present for a varying number of years prior to operation. The longest period was twelve years, and the shortest period was five days. Meningitis had occurred in 6 patients, and in 3 patients remissions had also taken place. Three of these patients had undergone operation previously for rhinorrhea, unilateral craniotomy having been employed, with attempts at plastic closure of a fistulous opening. In 7 of the 8 cases operation

*Cerebrospinal Rhinorrhea (26 Cases)*

| | Sex | | Sinuses | | |
|---|---|---|---|---|---|
| Origin | Males | Females | Frontal | Ethmoid | Frontal and Ethmoid |
| Traumatic | 4 | 4 | 2 | 3 | 3 |
| Spontaneous (nasal meningoceles) | 6 | 12 | 0 | 18 | 0 |
| Total | 10 | 16 | 2 | 21 | 3 |

was performed by one of us (Adson) and in the other case by Dr. G. S. Baker. The longest postoperative interval has been nine years. All 8 patients were cured. There were no deaths.

Spontaneous cerebrospinal rhinorrhea occurred in 18 cases in which operation was performed. Single fistulous openings were observed in 10 cases. Two or more fistulous openings were observed in 8 cases. Nasal polyps were observed by our otolaryngologists in 11 cases. Operation on nasal polyps (nasal meningoceles) had been performed at the Mayo Clinic or elsewhere in 9 cases. Our roentgenologists observed defects in the cribriform plate, with the presence of a mass in 3 cases. Dr. Hallberg, of the Section on Otolaryngology and Rhinology, permits me to quote him in stating the following:

I have seen 2 such patients. One was a 7 year old girl who had had a meningocele in the right side of her nose. The other patient was a man in his forties who had a meningocele in the left side of his nose. Both of these meningoceles had a whitish look and made their appearance in the nose between the septum and the middle turbinate. In this way they are differentiated from ordinary polyps, most of which originate in the middle meatus, that is, between

the middle turbinate and the lateral wall of the nose. If a meningocele in the nose is ruptured or torn, fluid can be seen trickling out of it by having the patient bend over. In most of the cases which we encounter cerebrospinal rhinorrhea comes on after intranasal surgical procedures, more often after removal of polyps than after any other type of operation. I do not feel that it is caused by trauma alone. I would not be surprised if, in some of them, meningoceles were mixed up in the polyp formation.

In further reviewing the 18 cases of spontaneous cerebrospinal rhinorrhea in which operation was performed, we learned that 12 of the patients were female and 6 were male. Two of the patients were children who had suffered from rhinorrhea from birth. In 1 case cerebrospinal fluid had continued to drain for fourteen years. In 3 cases fluid had drained for more than ten years. Remissions had occurred in 3 cases. Meningitis had occurred in 3 cases. In 2 patients previous attempts at repair had been made.

In addition, 3 patients who did not undergo surgical procedures have consulted us for cerebrospinal rhinorrhea. One patient was a woman, 48 years of age, who had suffered from cerebrospinal rhinorrhea for a period of nine years. She had had three attacks of meningitis, was admitted during her fourth attack and died two hours after admission. The second patient, a woman 40 years of age, gave a history of intermittent attacks of cerebrospinal rhinorrhea. After her examination had been completed, she was admitted to the hospital and was being prepared for surgical treatment with chemotherapy when the nasal drip ceased. Surgical intervention was postponed, and on recent examination we learned that she has not had a recurrence of her previous trouble with the exception of one morning when a few drops of clear fluid fell from her nose. This emphasizes the point that in an occasional case spontaneous recovery may take place. The patient made an interesting statement which was significant. She stated that prior to her initial admission to the clinic she had observed that the nasal drip was always greater when she was excited or had lost sleep. The third patient, a woman 40 years of age, complained of symptoms similar to those of which patients complain when cerebrospinal rhinorrhea is present. This patient was found to have what otolaryngologists like to refer to as an "allergic nose." No nasal polyps were demonstrable. One cubic centimeter of indigo carmine, which was introduced into the subarachnoid space by cisternal puncture, failed to appear in the nasal discharge; therefore, surgical intervention was not advised.

*Surgical Results.*—In 26 cases operation by the technic previously described by one of us (Adson) has been performed for cerebrospinal rhinorrhea. In 8 of these the rhinorrhea was of traumatic origin. All 8 patients were cured. In 18 cases cerebrospinal rhinorrhea developed spontaneously without a history of injury. Fourteen patients have been

cured. In 3 cases the operation was a failure. In 2 of the cases of failure reoperation has been performed without success. The results in the fourth case are equivocal since the patient has gone for many months without the appearance of cerebrospinal fluid in the nose and when it does appear only a few drops occur.

In regard to these failures, it is possible that the bone wax may have been dislodged as the result of sneezing or coughing which disturbed the sealed-off process effected at the time of surgical intervention. It is also possible that the wax had not been properly molded into the defect in the cribriform plate owing to the fact that soft tissue, the meninges, which extended through the cribriform plate, was not incised below the level of the bone. In 1 instance in which reoperation was performed it became apparent that there were several fistulous openings through the cribriform plate which had been overlooked at the primary operation.

## SUMMARY

Cerebrospinal rhinorrhea due to trauma (fractures) frequently ceases spontaneously; therefore, it is wise to wait for a period of eight weeks before correcting the situation. Palliative measures are rarely effective. Only in an occasional case will the cerebrospinal fluid cease to drain after application of these measures.

Nasal polyps fairly frequently are nasal meningoceles. These may break and drain cerebrospinal fluid spontaneously but are sure to drain if resected; however, there is no doubt that a small meningocele may be associated with nasal polyps. Meningitis is a serious complication of cerebrospinal rhinorrhea, and this is especially true in the presence of acute infections of the nose, throat, tonsils and sinuses.

Administration of penicillin and the sulfonamide compounds reduces the hazard of meningitis and serves as a protective measure in the preoperative and postoperative periods.

Attempts at repair of fistulous openings through a unilateral frontal craniotomy have not been entirely satisfactory. This is especially true when the lesions are located in the cribriform plate, since they are frequently multiple and may occur on both sides of the septum.

The best results are obtained by bilateral frontal craniotomy, with adequate exposure of the cribriform plate before closure of the defects in the cribriform plate and the meningocele fistulous openings.

In 26 patients cerebrospinal rhinorrhea was relieved. Twenty-two patients were cured. There was 1 equivocal result, 3 failures and no deaths.

# PSEUDOHEMOPHILIA OR CHRONIC THROMBASTHENIA

CHARLES W. McLAUGHLIN Jr., M.D.
OMAHA

THE CONDITIONS under discussion are those which have been called in the literature "pseudohemophilia or chronic hereditary thrombasthenia." These two dyscrasias were originally described nearly forty years ago, but it has only been in recent years that a clear differentiation has been drawn between the two entities and their characteristics more generally understood.

Glantzmann [1] in 1918 described a new diathesis occurring in both sexes characterized by bleeding of a purpuric type with hemorrhage from the mucous membranes and beneath the skin. In the original series the bleeding time, clotting time and platelet count were all reported to be normal. The clot retraction was delayed or absent. To this condition he gave the name "chronic hereditary hemorrhagic thrombasthenia" since he considered the platelets to be qualitatively defective. Subsequently cases considered to be in this group were reported by Van der Zande,[2] Rosenthal,[3] Little and Ayres,[4] Roskam [5] and Minot.[6] A careful review of the case reports, however, discloses that in a number of instances the findings in the blood originally described by Glantzmann were not present. In several of the patients whose condition was classified as "chronic hereditary thrombasthenia" the bleeding time was prolonged and clot retraction was found to be normal. Quick [7] has observed that in the period since Glantzmann's

Read at the Fifty-Sixth Annual Meeting of the Western Surgical Association, St. Louis, Dec. 2, 1948.

1. Glantzmann, E.: Hereditäre hämmorrhagische Thrombasthenie, Jahrb. f. Kinderh. **88**:1, 1918.

2. Van der Zande, F.: Pseudohemophilia, Nederl. tijdschr. v. geneesk. **1**: 544, 1923.

3. Rosenthal, N.: Hemorrhagic Diatheses—Chronic Hereditary Thrombasthenia, in Downey, H.: Handbook of Hematology, New York, Paul B. Hoeber, Inc., 1938, vol. 1, p. 523.

4. Little, W. D., and Ayres, W. W.: Hemorrhagic Disease: Familial Bleeding Tendency of Unusual Type with Splenomegaly Affecting and Transmitted by Both Males and Females, J. A. M. A. **91**:1251 (Oct. 27) 1928.

5. Roskam, J.: Purpuras hemorragiques et thrombopenia, Sang **3**:497, 1929.

6. Minot, G. R.: A Familial Hemorrhagic Condition Associated with Prolongation of the Bleeding Time, Am. J. M. Sc. **175**:301, 1928.

7. Quick, A. J.: The Hemorrhagic Diseases and Physiology of Hemostasis, Springfield, Ill., Charles C Thomas, Publisher, 1942.

initial report there are few cases in the literature which correspond to his original description. He further concluded that it is difficult to accept the possibility that clot retraction can be defective when the platelet count, bleeding time and clotting time are all normal. While such a condition associated with abnormal bleeding due to faulty clot retraction may exist, it must be extremely rare, and the diagnosis should be made only when the specific requirements are met.

In 1926 von Willebrand [8] described a family with a definite bleeding tendency in which the blood picture was characterized by a prolonged bleeding time with normal or slightly increased platelet count, a normal coagulation time, normal clot retraction and a negative reaction to the tourniquet test. This condition he called "pseudohemophilia." The original group were all found living on an island in the Baltic Sea where the disease was predominately observed in females, but subsequent studies have shown the sex incidence to be about equally divided. It was definitely proved, however, that the disease is transmitted by both sexes.

Estren, Midal and Dameshek [9] have collected 62 cases from the literature since 1910 which they consider definite examples of pseudohemophilia. In each of these there was a history of excessive bleeding and a definitely increased bleeding time in the presence of a normal platelet count and usually a normal coagulation time. A definite family history of bleeding was obtainable in from 50 to 75 per cent of their series.

Pseudohemophilia usually develops in infancy but may be latent until adult life. In general, the bleeding diathesis tends to diminish in severity as the person grows older, although the bleeding time usually remains prolonged throughout life. The severity of the bleeding tendency in a given subject is difficult to estimate and may vary strikingly from time to time in the same person. Perkins [10] has pointed out that the bleeding time in 1 of his cases fluctuated from eight minutes to an hour and twenty minutes in a few days' time by the same technic and varied during the same period in different sites of the body. Similar variations were observed in 1 of our cases.

Estren and others have recorded the common bleeding sites in their collected series as the nose in 75 per cent of the cases, the skin in 70 per cent and the gums in 25 per cent; the bleeding was of dental origin in 20 per cent. Bleeding from the gastrointestinal and urologic tracts and into the joints was much less frequently observed. Typical

8. von Willebrand, E. A.: Hereditare pseudohemofili, Finska läk.-sällsk. handl. **68**:87, 1926.

9. Estren, S.; Midal, L. S., and Dameshek, W.: Pseudohemophilia, Blood **1**: 504, 1946.

10. Perkins, W.: Pseudohemophilia, Blood **1**:497, 1946.

petechiae were distinctly unusual, but extensive subcutaneous hemorrhage with or without trauma and a tendency to bleed easily after minor injuries were commonly observed.

The conditions most frequently to be confused with pseudohemophilia are true hemophilia, thrombocytopenic purpura and chronic hereditary thrombasthenia (tables 1 and 2). Idiopathic hypoprothrombinemia may present almost identical clinical and laboratory findings except that the prothrombin time in this condition is persistently low and resistant to all therapy.[11]

TABLE 1.—*Hemorrhagic Diseases—Differential Diagnosis*

| | Pseudohemophilia | Hemophilia | Essential Purpura | Chronic Hereditary Thrombasthenia |
|---|---|---|---|---|
| Hereditary | Dominant | Recessive | ..... | Dominant |
| Transmission | Male and female | Female | ..... | Male and female |
| Bleeding tendency time after injury | Immediately | Usually delayed | Immediately | Immediately |
| Petechial | Uncommon | Absent | Common | Common |
| Ecchymotic | Common | Present | Extremely common | Common |
| Mucosal | Severe | Uncommon | Severe | Severe |
| Hemarthrosis | Uncommon | Common | Absent | Uncommon |

TABLE 2.—*Blood Tests in the Differential Diagnosis of Hemorrhagic Diseases*

| | Pseudohemophilia | Hemophilia | Essential Purpura | Chronic Hereditary Thrombasthenia |
|---|---|---|---|---|
| Bleeding time | Prolonged | Normal | Prolonged | Usually normal |
| Coagulation time | Normal | Prolonged | Normal | Normal |
| Platelet count | Often increased | Normal | Decreased | Normal or low |
| Tourniquet test | Positive or negative | Negative | Positive | Usually positive |
| Clot retraction | Usually normal | Normal | Absent | Prolonged |

The underlying pathologic process in pseudohemophilia is as yet not clearly understood but probably lies in the vessels themselves. MacFarlane [12] has shown by microscopic studies of the nail beds of patients with this condition that there exists distorted and bizarre patterns with little or no response to injury. Subsequently Perkins repeated MacFarlane's work and was able to confirm the presence of abnormal capillaries which did not respond to ordinary stimuli. Dameshek [13]

11. Rhoads, J. E., and Fitz-Hugh, T., Jr.: Idiopathic Hypoprothrombinemia—An Apparently Unrecorded Condition, Am. J. M. Sc. **202**:662, 1941.

12. MacFarlane, R. G.: Critical Review: Mechanism of Hemostasis, Quart. J. Med. **10**:1, 1941.

13. Dameshek, W.: Medical Progress—Hemorrhagic Diseases, New England J. Med. **230**:542, 1944.

attempted to repeat MacFarlane's studies, without definite success. He concluded that the abnormal mechanism in this condition must as yet be considered unknown.

That the platelets are normal in true pseudohemophilia apparently has been adequately demonstrated. Buckman [14] has shown that blood from a patient with pseudohemophilia added to blood from a hemophilic patient causes rapid coagulation, as will blood from a normal donor.

There is no satisfactory or specific therapy for pseudohemophilia to date. Calcium and vitamin C have been used without benefit to the bleeding tendency. Vitamin K is without benefit since prothrombin levels are normal in cases of proved pseudohemophilia. Irradiation over the spleen has been tried, with little success. Splenectomy has been attempted in 4 patients, 1 of whom succumbed four hours after operation with uncontrollable hemorrhage. Two other patients evidenced no clinical improvement, and the fourth patient, while showing slight general benefit, retained the same bleeding tendency. Snake venom has been suggested as a therapeutic agent since it acts as thrombokinase converting prothrombin into thrombin in the presence of calcium. Venom was given a prolonged trial in 1 of our cases, with little evident effect. Transfusions of whole blood have a variable influence on active hemorrhage in pseudohemophilia. While in some instances they seem to allay the tendency to bleed for a time, the major benefit is apparently through replacement of blood lost.

It is well established from ample experience that elective surgical intervention in these patients is to be undertaken only after serious consideration. Hemorrhage may be most difficult to control or fatal in certain instances. Death from persistent bleeding following minor injuries, however, is rare, only 2 cases being found in the literature in the past thirty years. It may, therefore, be concluded that the prognosis for life is excellent, and although the bleeding tendency persists, it may become less marked as the patient grows older. It is extremely important to recognize the tendency from the standpoint of prophylaxis.

We have recently had occasion to observe 2 children who demonstrated the characteristic findings which have been classified under the name of "pseudohemophilia." The 2 cases are here reported.

CASE 1.—D. K., a white male child 18 months of age, was admitted to the hospital on June 19, 1946, five days after sustaining a dog bite of the face. The family history did not reveal any instances of a bleeding diathesis. The patient, however, had been observed to bleed easily from minor trauma since birth. The child had been attacked by a dog five days before admission, sustaining a deep

14. Buckman, T. E.: Atypical Pathologic Hemorrhage in Early Life, Am. J. M. Sc. **175**:307, 1928.

fang bite centrally placed in the right cheek. Initial bleeding was profuse, and after local cleansing primary suture was done by the family physician. The wound continued to ooze, and the child was referred to the hospital for further care.

On admission the child was seen to be well nourished but pale and apprehensive. There was a 1 cm. wound centrally placed in the right cheek closed by three deep sutures. There was a large blood clot loosely adherent to the suture line, with fresh oozing from beneath the clot. There was no penetration of the wound into the mouth. The liver and spleen were not palpable, and the regional nodes were not enlarged. There were several areas of subcutaneous hemorrhage noted over the extremities. The reaction to the tourniquet test was negative.

Initial laboratory studies showed nothing striking except the evidence of considerable loss of blood. The hemoglobin content was 6.6 Gm., the red blood cell count was 2,270,000 and the white blood cell count was 6,000. A differential count showed 13 per cent segmented cells, 2 per cent staff cells, 74 per cent lymphocytes, 5 per cent monocytes and 6 per cent eosinophils. The bleeding time was seven minutes, the clotting time was seven minutes and the platelets numbered 232,000.

On the day of admission 250 cc. of citrated blood was given after exposure of the left long saphenous vein. It was noted that this small wound did not bleed abnormally. The following day the wound on the face was still oozing, and on the third day after admission it was explored under open drop ether anesthesia. The original sutures and the old blood clot were removed. The wound was seen to be 1 cm. long and 1 cm. deep and was not grossly infected. One vein in the soft tissues seemed to be the source of bleeding. This was ligated, and the wound was closed with three deeply placed sutures with hemostatic gauze tied over the line of closure.

The wound continued to bleed intermittently during the next two days, and it was again explored under general anesthesia three days later. A piece of necrotic tissue was removed, but no specific bleeding points could be identified for ligation. Three pieces of gelfoam® were buried in the wound and dead space obliterated by fine surgical gut suture. The skin was closed with silk and a collodion dressing applied.

After this procedure there was little further bleeding, and the child was dismissed on the eleventh day in the hospital with the wound healing satisfactorily.

On Sept. 7, 1946, approximately two months later, the child was readmitted to the hospital. A week before admission he had been bitten by a mosquito on the shoulder, and the area was scratched, with resultant persistent bleeding which had continued. In the interval since discharge the face had become completely healed, with minimum scarring, but areas of spontaneous subcutaneous hemorrhage had been frequently noted.

On this admission there were numerous small areas of subcutaneous hemorrhage visible over the trunk. The left leg was markedly swollen from the knee to the ankle because of massive hemorrhage into this extremity, which had developed spontaneously three days before admission. Laboratory data showed the hemoglobin content 7.9 Gm., red blood cell count 3,850,000, white blood cell count 16,500, with 60 per cent segmented cells, 8 per cent staff cells, 28 per cent lymphocytes and 4 per cent monocytes. The ear was stuck for a blood specimen and continued to bleed for twenty-four hours. The bleeding time continued to be somewhere between one and twenty-four hours. The clotting time by the test tube method was ten minutes. The clot retracted, but this was slightly delayed. The platelets numbered 200,000. The child was given 250 cc. of citrated blood through an exposed saphenous vein, again there being no abnormal bleeding from this small surgical incision.

Bleeding from the mosquito bite ceased after the transfusion and did not recur during the twenty day stay in the hospital. The areas of subcutaneous hemorrhage faded, and the massive hemorrhage into the left leg gradually absorbed, with no involvement of the joints. The spleen progressively enlarged during observation in the hospital until it was palpable 3 fingerbreadths below the left costal margin. It then receded and was not palpable at the time of dismissal. The child was dismissed on snake venom, 0.2 cc. to be administered twice weekly.

In June 1948, two years after the first admission, he was again admitted for further study. During this period he had grown and developed normally. However, he had frequently evidenced large areas of subcutaneous hemorrhage without known trauma and had also had massive hemorrhages into the legs and thighs and over the scapulas. These had always resolved without residual defect. There had never been any gastrointestinal bleeding, and the spleen had never again been palpable.

On this admission, many areas of subcutaneous hemorrhage were noted over the buttocks and over both lower extremities. The reaction to the tourniquet test was again negative.

TABLE 3.—*Laboratory Data on Baby D. K., Aged 18 Months**

| | June 19, 1946 † | June 22, 1946 †‡ | Sept. 7, 1946 † | Sept. 27, 1946 | June 2, 1948 |
|---|---|---|---|---|---|
| Hemoglobin, Gm. | 6.6 | 8.9 | 7.9 | 9.4 | 13 |
| Red blood cells | 3,270,000 | 3,410,000 | 3,850,000 | 3,100,000 | 4,950,000 |
| White blood cells | 6,000 | 7,500 | 13,500 | 8,000 | 5,400 |
| Differential count | Normal | Normal | Normal | Normal | Normal |
| Bleeding time | 7 min. | 6 min. | 30 + min. | 1 to 36 hr. | 18 + min. |
| Coagulation time, min. | 5 | 10 | 10 | 10 | 12 |
| Platelets | 232,000 | ..... | ..... | 200,000 | 90,000 to 120,000 |

* The prothrombin time, clot retraction and bone marrow were normal.
† Transfusion of blood given.
‡ Sutures applied.

An examination of bone marrow was carried out by needle puncture on June 2, 1948. The bone marrow was reported to be moderately hyperplastic, with megakaryocytes present. It is noteworthy that the sternal puncture wound bled for seventy-two hours and a needle puncture wound in the heel at the same time for a blood specimen bled for twenty-four hours. The hemoglobin content was 13 Gm., the red blood cell count 4,950,000 and the white blood cell count 5,400, the segmented forms numbering 56, staff forms 8, lymphocytes 46, monocytes 2 and eosinophils 2. The clotting time was twelve to eighteen minutes. The clot retraction began in thirty minutes and was complete in five hours. The platelets were reported to number 130,000 and on succeeding days 90,000, 82,000 and 84,000. This decrease was noted only at the time of this admission to the hospital, all previous counts being between 175,000 and 250,000. The child had been given snake venom during the two year period, and the use of this was discontinued at the time of the third admission. The hemorrhagic tendency still persists, but general development has been normal to date (table 3).

The final diagnosis was pseudohemophilia. Essential thrombocytopenic purpura was seriously considered for a time, particularly when the platelets were definitely reduced in number. However, the striking splenomegaly observed for a few days in this child is practically never seen in essential purpura.

CASE 2.—K. H., a white male infant 1 year of age, was admitted to the hospital on Aug. 4, 1947, with persistent bleeding from a small wound in the tongue. Two days before admission the child fell at play, biting the lateral edge of the tongue. The area bled profusely and was controlled intermittently by pressure during the first twenty-four hours. Subsequently it began to bleed continuously, and the child was referred to the hospital for further study.

Physical examination on admission disclosed a rather fat child of 1 year who was pale and had obviously lost considerable blood. Inspection of the oral cavity demonstrated the presence of fresh blood, with a large blood clot over the left lateral edge of the tongue. Removal of the clot disclosed a small deep puncture wound 3 mm. in length which did not go completely through the tongue. The spleen was not palpable, and there were no enlarged lymph nodes. Numerous areas of subcutaneous hemorrhage varying in size were visible over the trunk and extremities.

The only instance of a bleeding diathesis in the family involved an older brother. Four years before a male baby 6 months of age in the same family sustained a small puncture wound of the tongue. Bleeding was profuse and persisted, and the child died from loss of blood in a hospital while arrangements for a transfusion were being made. No blood studies were available.

The initial laboratory studies showed a hemoglobin content of 56 per cent, a red blood cell count of 3,500,000 and a white blood cell count of 12,000. The differential count was normal. The bleeding time was reported to be thirty minutes, the clotting time twelve minutes and the platelets 250,000.

The following day oozing had persisted, and under general anesthesia the wound in the tongue was carefully inspected and closed with two deeply placed silk sutures tied over a piece of gelfoam.* A transfusion of 125 cc. of blood was given. The wound had remained dry the following day, and the bleeding time was found to be twenty minutes and the clotting time five minutes. The child was dismissed under the further care of the referring physician.

On August 9, three days after dismissal, the child was readmitted with further bleeding from the tongue. This was apparently induced by chewing on a plastic toy when his arms were temporarily freed from the prescribed restraints. The bleeding persisted, and the following day the child was given 150 cc. of blood and the tongue again inspected under divinyl oxide anesthesia. Three silk sutures were introduced deep to the puncture wound and tied over a piece of gelfoam.*

Bleeding was entirely controlled for three days but recurred again on August 13, when a third transfusion of 175 cc. of blood was administered. The tongue was again exposed under general anesthesia, and while the original wound appeared almost healed, persistent oozing was noted from the silk suture holes. These were closed with deeply placed sutures over gelfoam.* It was of interest that no abnormal bleeding occurred from the small cut-down incision used to expose the long saphenous vein for transfusion.

An examination of bone marrow was made on August 13 and was reported as follows: Cytologic examination of marrow elements reveals a normal distribution of the myeloid-erythroid series. Various stages of maturation of red cells are present, varying from the occasional megaloblast to the mature red cells. No abnormal number of myeloblasts are present. The megakaryocytes appear normal in number and show normal pseudopodia in which platelets are present . Abnormal forms such as sharply defined cytoplasmic borders with a loss of granularity of the cystoplasm and vacuolization of the cystoplasm are present. The diagnosis is normal bone marrow.

Three days later, on August 16, the bleeding recurred in a profuse form. A fourth transfusion of 150 cc. of blood was given and the bleeding site again closed with mattress sutures tied over gelfoam® impregnated in topical thrombin.

On August 17, only eighteen hours after the fourth attempt at local control of bleeding by suture, the wound was again observed to be oozing briskly. A transfusion (the fifth) of 150 cc. of blood was given, and after considerable discussion the left external carotid artery was exposed in the neck under general anesthesia. The external carotid artery was doubly ligated with silk sutures just above the superior thyroid branch, and this vessel was ligated in addition as a precaution. No excessive bleeding occurred from this incision in the neck. The previously placed sutures in the tongue were removed, and no further oozing was observed.

On August 21, four days after the carotid ligation, there was only a little oozing noted from the tongue. The incision in the neck appeared normal. Two days later, sudden profuse hemorrhage from the tongue again occurred and persisted for two hours. The child was given a sixth transfusion of 150 cc. of blood, but further local suture was not immediately undertaken.

Bleeding stopped spontaneously and did not recur for five days, when there was again profuse sudden hemorrhage from the tongue. The child was given a seventh

TABLE 4.—*Laboratory Data on Baby K. H., Aged 1 Year* *

| | Aug. 4, 1947 †‡ | Aug. 11, 1947 †‡ | Aug. 14, 1947 †‡ | Aug. 25, 1947 †§ | Aug. 28, 1947 †‡ | May 18, 1948 |
|---|---|---|---|---|---|---|
| Hemoglobin, per cent..... | 53 | 81 | 71 | 35 | 45 | 60 |
| Red blood cells..... | 3,480,000 | 3,940,000 | 4,010,000 | 2,350,000 | 2,470,000 | 2,980,000 |
| White blood cells..... | 12,000 | 11,600 | 11,700 | ..... | 8,950 | 8,000 |
| Differential count..... | Normal | Normal | Normal | ..... | Normal | Normal |
| Bleeding time, min..... | 30 | 20 | ..... | ..... | ..... | 6½ |
| Coagulation time, min.... | 5 | 3.5 | ..... | ..... | ..... | 2 |
| Platelets ..... | 294,000 | ..... | ..... | ..... | ..... | 342,000 |

* The prothrombin time, clot retraction and bone marrow were normal.
† Transfusion of blood given.
‡ Sutures applied.
§ Carotid ligation.

transfusion of 240 cc. of blood and the tongue exposed under divinyl oxide anesthesia. The original wound on the lateral dorsum appeared dry and practically healed. There were two puncture wounds on the lateral edge of the tongue with raised edges appearing much like old suture wounds, and from these brisk bleeding was noted. The holes were carefully explored, and no foreign material was found. The tongue felt entirely normal to palpation. The puncture wounds were closed with 000 chromic surgical gut sutures, with prompt control of the bleeding. There was no further hemorrhage, and the child was dismissed from the hospital on August 30 with the wound dry.

He was readmitted nine months later, on May 17, 1948. For one week prior to admission there had been persistent bleeding from the gingival mucosa over an erupting upper right molar tooth. On this admission numerous areas of subcutaneous hemorrhage were again noted, but the spleen and regional nodes were not palpable. During the preceding months the child had bled freely on a number of occasions after minor cuts and scratches, but in every instance this had stopped spontaneously. On this admission the bleeding time was six and a half minutes, and the coagulation time two minutes by the capillary tube method, and clot retraction began in one hour and was complete in three hours, with a firm clot. The prothrombin time was 16.5 seconds, or 105 per cent of normal. The hemoglobin

content was 7.5 Gm., the red blood cell count 2,980,000 and the white blood cell count 8,000. The differential count was normal. The platelets numbered 342,000.

Three days after admission the child was given 180 cc. of blood because of persistent oozing, after which the bleeding ceased. A sample of the baby's venous blood was mixed with his saliva and the coagulation time checked against a control specimen from the patient, without significant difference in the result.

At the time of dismissal the gums were no longer bleeding and the hemoglobin content was 9.5 Gm., with a red blood cell count of 3,010,000 (table 4).

The diagnosis was pseudohemophilia. While the bleeding time was not so strikingly prolonged in this case, it was increased at times, and all other laboratory data were in keeping with this diagnosis.

The family history was of unusual interest in that an older brother died with a similar history. While data are inadequate to make a diagnosis, they are certainly suggestive and noteworthy since only two previously reported cases of fatal hemorrhage have been found in the literature.

## SUMMARY AND CONCLUSIONS

1. Pseudohemophilia and chronic hereditary thrombasthenia are probably two distinct entities.

2. Both conditions present themselves as hemorrhagic diathesis, usually familial in type but not sex linked.

3. Pseudohemophilia is a more clearly established condition in which the principal diagnostic feature is a persistently prolonged bleeding time.

4. Two cases classified as instances of pseudohemophilia are reported.

5. There is no specific therapy for pseudohemophilia, but the various means of controlling hemorrhage have been discussed.

6. Pseudohemophilia is rarely fatal, the bleeding tendency usually becoming less marked with advancing years. Surgical treatment should be avoided unless absolutely required.

## DISCUSSION

DR. GEORGE CURTIS, Columbus, Ohio: V. P., a baby boy of 10 months, was admitted to the University Hospital on Jan. 6, 1944, presenting the clinical problem of "a bleeding tendency." He had had recurrent crops of subcutaneous ecchymoses, beginning at the age of 7 months and continuing up to the time of admission. There were no other obvious signs or symptoms in this well developed, well nourished and otherwise apparently normal infant. No other bleeding had been noted, and there was no familial history of any hemorrhagic diathesis, including hemophilia. The patient had 2 brothers and 1 sister, who were normal, as were the parents.

On admission, the patient presented red cheeks and mucosae and only one ecchymotic area, about 1 cm. in diameter, on the left knee. The general physical examination was essentially noncontributory. There was no lymphadenopathy. The liver and spleen were not palpable. The temperature was normal. The baby's disposition was excellent, and the diet was well taken. There was no blood in the urine.

The blood count on admission was: white blood cells, 23,300; red blood cells, 5,190,000; hemoglobin content, 9 Gm.; blood platelets 290,000 per cubic millimeter, and cell volume, 41 per cent. The sedimentation rate was normal. The bleeding time was three minutes. The coagulation time (Howells' method) was twenty-two minutes. Clot retraction was normal.

The following day, January 7, the white blood cells numbered 13,450, with 28 per cent granulocytes, 66 per cent lymphocytes and 6 per cent monocytes. The red blood cell count was 4,370,000, with 1.4 per cent reticulocytes, 9 Gm. of hemoglobin and over 1,000,000 platelets per cubic millimeter.

Forty-eight hours after admission (the morning of January 8) we were greatly disturbed to find the little fellow pale, inactive and unresponsive. The red blood cell count had fallen to 1,830,000, the hemoglobin to 5.5 Gm. and the platelets to 231,000. The white blood cell count was 22,200, with 84 per cent polymorphonuclear cells, 15 per cent lymphocytes and 1 per cent monocytes. During the succeeding seven hours the red cells fell to 1,190,000 and the hemoglobin to 4 Gm.; the baby appeared moribund. There was no frank icterus. Neither the spleen nor the liver was palpable. There was no evidence of external loss of blood. There was evidence of extensive hematoma formation in the left leg, however, not apparently sufficient to cause such a precipitous fall of the red cells.

Studies of erythrocyte fragility at this time were suggestive. One hundred cubic centimeters of blood was immediately started into the marrow of the left tibia. The red blood cell count rose to 1,900,000. This was followed by a second transfusion, after which the count rose to 3,060,000 and the hemoglobin content to 7.8 Gm. The prothrombin time was 107 per cent. There was no evidence of external hemorrhage, although hematoma formation in the left thigh was obvious. There was no significant thrombocytopenia at this or any other time.

Subsequent studies of both serum and cells indicated an inherent increased fragility of the red cells, regarded as suggestive of hemolytic icterus of the congenital type.

Sternal aspiration revealed a grossly hyperplastic bone marrow, with the myelocytes normal, as well as a definite hyperplasia of the erythrocyte cells, most of which were in the normoblastic stage. There were many megakaryocytes including many young mononucleated forms such as characterize the splenic type of thrombocytopenic purpura.

Eleven days after admission (January 17) it was possible to palpate an enlarged spleen for the first time,

Thus, accumulating evidence appeared to point toward a syndrome consisting of splenic hemolysis together with a secondary intermittent thrombocytolysis, and the factor of hypersplenism came under consideration. As a consequence splenectomy was considered and then advised, in the double hope of preventing a recurrence of any erythroclastic activity and the purpuric manifestations. After another preoperative blood transfusion a 27 Gm. spleen, measuring 6.5 by 2 by 3.2 cm., was removed on January 20 without any difficulty and with no immediate complication. Fortunately, the surgical anatomy was such that only three intra-abdominal ligatures were necessary, one on the ligament to the diaphragm, along the cardia, one on the pedicle and one on the pedicle of an accessory spleen removed from along the greater curvature of the stomach.

During the procedure the peritoneum did not become freckled with purpuric spots, as it ordinarily does during splenectomy for thrombopenic purpura. The wound, however, was soggy, and the oozing continued even after the splenectomy, during closure.

Supravital staining of scrapings of the splenic pulp revealed a great increase in highly phagocytic clasmatocytes, as well as numerous sequestered blood platelets. Lymphopoiesis was normal, as was other cellular activity. The microscopic sections revealed hyperplasia of the reticulum cells.

Later in the day it was noted that the dressings were stained with fresh blood. On closer inspection, two points of oozing were discovered in the incision. This was superficial. At no time was there any evidence of internal hemorrhage.

The total red blood cell count rose from 3,500,000 preoperatively to 5,000,000 postoperatively and the hemoglobin from 8.8 to 12.5 Gm. The spleen had been well massaged just previous to the splenectomy. The blood platelets rose to 1,355,000 immediately after operation. Later during the same day the platelets fell to 134,000 per cubic millimeter and the red blood cells to 2,810,000. The white blood cell count was 25,000, with 77 per cent granulocytes.

The steady oozing continued. Consequently, various methods were used to control the bleeding from the site of the incision, including repeated small blood transfusions. The wound was opened and resutured; however, there was no single vessel to ligate. Pressure was helpful, but bleeding resumed when it was released. Adhesive strapping was helpful. Gelfoam® was not available. Epinephrine in 1 to 1,000 dilution, applied locally, would stop the ooze for a while. The best result was obtained by the use of thrombin topically. The hemoglobin and red cells did not return to a critically low level even in these trying circumstances. Yet intermittent bleeding continued for three weeks, even though the capillary coagulation time, read from the blood as it collected in the oozing suture line, was normal. Infection of the wound did not occur. Sulfadiazine was used locally as well as generally.

While the prothrombin time and blood platelets fluctuated considerably, at no time were they sufficiently low to account for the bleeding, which occurred not only in the operative incision but also at times at the site of diagnostic needle puncture in the finger or lobe of the ear.

It is our present feeling that there exists, in addition to the known factors essential to blood coagulation, some other important element which was lacking in this little patient and that this deficiency probably persisted, even after splenectomy. This can be proved by the test of time together with further experience and investigation. In the meantime every precaution should be taken with this patient to prevent any unnecessary trauma which might result in restarting a hemorrhage so difficult to control. It is possible by repeated transfusions of fresh whole blood to control the boy's hemorrhagic tendency. If facilities are available for this lifesaving measure, there should be no great immediate danger.

On discharge (Feb. 13, 1944) the white blood cell count was 26,000, the red blood cell count 3,540,000, the reticulocytes 4.4 per cent, the blood platelets 969,000 and the hemoglobin 9.3 Gm. Removal of the enlarged spleen should prevent any further tendency toward splenic hemolysis and the splenic destruction of platelets such as occurs in thrombopenic purpura. Generous amounts of vitamin C and vitamin K appear to be indicated, to insure against any deficiencies of these factors in the blood coagulation mechanism.

Splenectomy is contraindicated for the enlarged spleen occurring in the course of pseudohemophilia.

DR. CHARLES W. McLAUGHLIN JR., Omaha: One point which I should like to stress is that the bleeding time, which is the one essential in making an accurate diagnosis, varies strikingly in these patients. In Dr. Curtis' case it was three minutes at the time the patient had intractable bleeding after splenectomy. One must make repeated determinations of the bleeding time from different sites before a final diagnosis is made.

# SURGICAL TREATMENT OF BENIGN AND SECONDARILY MALIGNANT TUMORS OF THE ESOPHAGUS

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ROCHESTER, MINN.

THE OCCURRENCE of benign tumors of the esophagus has been known since an early period of clinical diagnosis of lesions of the esophagus. The growths which were diagnosed at that time were of the pedunculated polypoid type and were recognized because of the periodic appearance of the tumor or its extrusion into the mouth of the patient. The treatment of these growths is probably the earliest of recorded surgical procedures for tumors of the esophagus.

Sussius (Susio) is credited by Minski [1] with one of the earliest necropsy descriptions (1559) of an esophageal polyp, which extended from the middle part of the esophagus to the cardia of the stomach, causing obstruction and death. Minski also credited to Dallas and Monroe one of the earliest attempts at surgical removal, which was carried out by them in 1763 on a 64 year old patient who, on vomiting, forced multiple pedunculated tumors into the mouth, causing severe laryngeal obstruction. Transoral removal of the growths was decided on, and a preliminary tracheotomy was done to prevent suffocation during the procedure. A portion of the pedunculated tumors was ligated with a snare and permitted to drop into the esophagus. The patient died two years later (April 1765) from starvation, the result of esophageal obstruction from continued growth of the remaining tumors.

Although the occurrence of benign tumors has been known for many years, they are relatively infrequent. Their subjective symptoms and clinical manifestations are often meager, a fact which often delays their clinical recognition. The only effective treatment for these growths is complete surgical removal, which often requires extensive operative procedures. Their surgical treatment has developed slowly because of the many problems involved. These problems are concerned not only with the establishment of a definite diagnosis but also with complex technical considerations of operative treatment.

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From the Division of Surgery, Mayo Clinic.

Read at the Fifty-Sixth Annual Meeting of the Western Surgical Association, St. Louis, Dec. 3, 1948.

1. Minski, P. R.: Zur Entwicklungsgeschichte und Klinik der Polypen und polypenähnlichen Gewächse des Rachens und der Speiseröhre, Deutsche Ztschr. f. Chir. **41**:513-587, 1895.

The development of roentgenologic and endoscopic methods of examination of the esophagus has made it possible to make an early diagnosis of the presence of a lesion and also to determine the location, extent and character of the lesion. The accurate information obtained from these methods of examination has been an important factor in the development of methods of treatment and is essential to the determination of the type of surgical treatment to be instituted.

The study of proved cases has shown that these tumors occur in different locations of the esophagus. The histologic examination of the growths shows that they arise from different tissues of the wall of the esophagus and that they may undergo malignant change.

In May 1944 I reported, before the American Association for Thoracic Surgery, 5 cases in which I had treated benign tumors of the esophagus surgically.[2] The purpose of the present paper is to report 6 additional cases in which I have operated since that time and to give a statistical statement of the clinical manifestations, surgical treatment and histologic observations in the entire series.

These benign tumors are of two general groups, the mucosal and the extramucosal or intramural tumors. The mucosal tumors are usually pedunculated and arise from the submucosa. They are of two types: lipofibromas, which have a single pedicle, and fibromyxomas, which may be multiple and have multiple pedicles. The extramucosal or intramural tumors arise from the muscle of the wall of the esophagus. They are usually of multicentric origin and present as multiple leiomyomas involving a considerable part of the wall of the esophagus or as leiomyomatosis involving the entire circumference of the esophageal wall, but occasionally there is a single circumscribed tumor. When the tumors are benign they do not involve the mucous membrane, but they may undergo malignant change and invade the mucosa, causing ulceration and hemorrhage.

The growth of benign tumors of the esophagus is usually slow, and they may attain considerable size without giving rise to subjective symptoms. This is particularly true of the intramural tumors, which rarely produce obstruction of the esophagus unless they attain great size. The common symptoms associated with these growths are dysphagia, substernal pain, regurgitation of food, cough and dyspnea. The pedunculated tumors are commonly associated with obstructive symptoms, and the diagnosis is often definitely established by regurgitation of the tumor into the mouth, which may be the first objective sign. Regurgi-

2. Harrington, S. W., and Moersch, H. J.: Surgical Treatment and Clinical Manifestations of Benign Tumors of Esophagus with Report of Seven Cases, J. Thoracic Surg. **13**:394-414 (Oct.) 1944.

tation of these growths into the nasopharynx can imperil life because of blocking of the trachea, which may result in strangulation.

The roentgenographic and roentgenoscopic examination of the esophagus is of great value in determining the presence of the growths. Esophagoscopic examination is of utmost importance in establishing a definite diagnosis, although at times it may be difficult to determine the site of origin as well as the type of growth because of the difficulty in obtaining a specimen for biopsy through the normal esophageal mucosa. The site of origin of the pedunculated tumors usually can be determined definitely.

The indications for surgical treatment of benign tumors are not as urgent or as imperative as for malignant lesions of the esophagus. They depend somewhat on the patient's general condition, the amount of difficulty the tumor is causing and the site of the tumor. Inasmuch as the growths may undergo malignant change, surgical treatment should be considered in all cases.

The surgical treatment of these tumors depends on whether the growth is submucous or pedunculated or is intramural and arises from the muscular coat of the esophagus. The pedunculated tumors may be more accessible and amenable to surgical treatment than the intramural tumors. If the pedunculated tumor is small and its origin is high at the introitus, it may be removed through the mouth with a snare and cautery. Larger tumors of this type require cervical esophagotomy. The tumors may attain such huge size as to require transthoracic esophagotomy. This approach also may be necessary if the pedicle of the tumor is situated low in the esophagus.

The indications for surgical treatment of intramural tumors are not as a rule as urgent as those for pedunculated tumors, because the former usually do not cause obstructive symptoms. The surgical treatment of these tumors often requires a much more extensive operative procedure than is necessary in cases of pedunculated tumors. The type of operative procedure to be carried out for intramural growths cannot be definitely determined by the clinical examinations, because of the difficulty in determining the type or extent of the growth. The accurate determination of the factors on which the indications for the type of operative procedure depend can be definitely made only at the time of operation. If the intramural tumors involve only a segment of the wall of the esophagus they may be removed by transthoracic and transpleural local excision of the tumor from the wall, which is then reconstructed. If the growth involves the entire esophageal wall, its removal will require transthoracic and transpleural resection of the esophagus and transdiaphragmatic esophagogastroanastomosis.

## REPORT OF CASES

The following is a summary of 6 cases in which I have operated since 1944; in 4 of these cases the tumors were benign intramural leiomyomas, and in the remaining 2 they were malignant leiomyosarcomas. In the subsequent section of the paper I shall include the 5 cases which I have previously reported, in 2 of which the tumors were pedunculated submucosal tumors and in 3 intramural leiomyomas. These 5 cases were reported before the American Association for Thoracic Surgery in May 1944.

CASE 1.—A man, aged 31 years, came to the Mayo Clinic because of the finding of a "tumor in the chest" by a routine roentgenologic examination of the thorax. He had had no subjective symptoms prior to the roentgenologic examination.

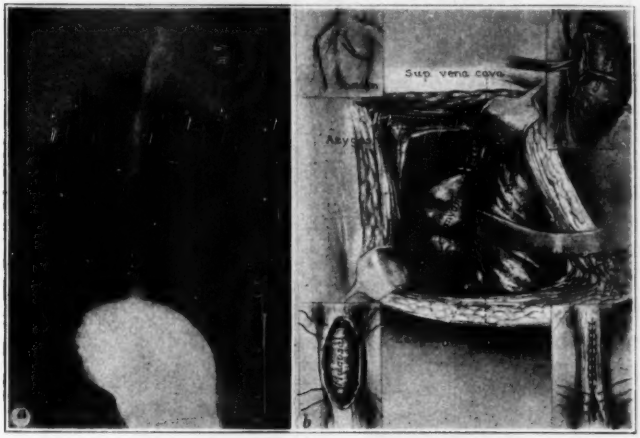



Fig. 1.—Intramural leiomyoma of middle third of the esophagus. Transpleural esophagotomy on the right side, with complete removal of tumor and reconstruction of the wall of the esophagus. *a*, rounded tumor of the right wall of the middle third of the esophagus partially obstructing its lumen on the patient's admission to the hospital; *b*, technic of complete excision of the tumor without opening the mucous membrane and complete reconstruction of the wall of the esophagus.

Three months prior to his admission to the clinic, after the tumor had been found, he had a nonproductive cough and a vague painful sensation in the anterior portion of the thorax.

The results of physical examination were negative. The roentgenogram of the thorax and esophagus revealed a rounded tumor mass displacing the esophagus to the right and posteriorly at the level of the carina (fig. 1 *a*). Because of the roentgenologic findings, an esophagoscopic examination was carried out, and a tumor was found involving the anterior and right lateral wall of the middle third of the esophagus. It appeared to be intramural.

A diagnosis was made of leiomyoma involving the anterior and right lateral wall of the middle third of the esophagus, and surgical intervention was advised.

Accordingly on July 8, 1944, transpleural esophagotomy, with complete resection of the tumor and reconstruction of the wall of the esophagus, was performed on the right side. The pleural cavity was entered by incision of the pleura through the posterior periosteum of the sixth rib, exposing a large tumor involving the wall in the middle third of the esophagus, just beneath the azygos vein (fig. 1 *b*).

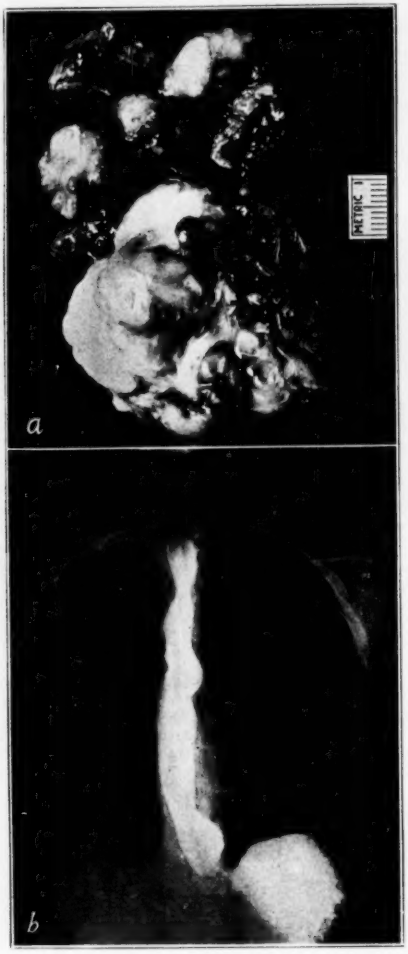



Fig. 2 (same case as in figure 1).—*a*, multicentric leiomyoma involving approximately two thirds of the circumference of the wall of the esophagus, measuring 5.5 by 5 cm. *b*, appearance on dismissal of the patient two months after operation and drainage of localized empyema. Esophagus is irregular at the site of reconstruction but otherwise normal.

It involved at least one half of the circumference of the right anterior and lateral muscular walls of the esophagus. The tumor, which was multicentric, was carefully dissected out of the wall of the esophagus without injury to the mucous membrane. The portion of the wall of the esophagus involved was completely destroyed by the tumor. This fact made it difficult to reconstruct the wall of the esophagus, but this was accomplished with three invaginating rows of silk sutures. The esophagus was considerably dilated at the site of the tumor, which made it possible to accomplish the reconstruction of the wall without obstructing its lumen. After invagination of the esophageal wall, the parietal pleura was accurately sutured for the reconstruction. The pleura attached to the lung was left open so as to drain the mediastinum. The tumor measured 5.5 by 5 by 2.5 cm. (fig. 2*a*). On microscopic study it was found to be a leiomyoma.

During the patient's postoperative course, empyema developed on the right side, for which an open operation with resection of two ribs was required. His sub-

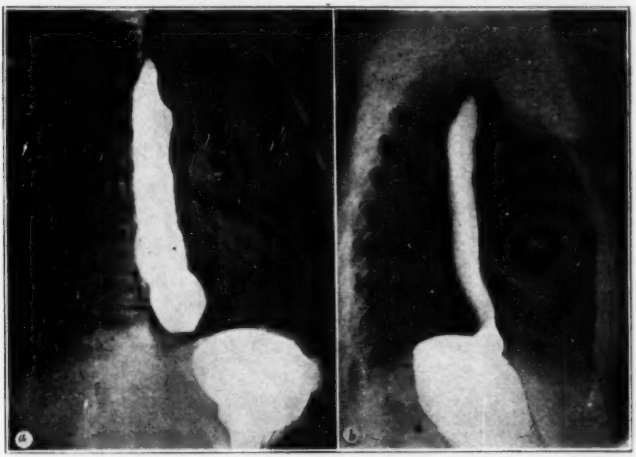


Fig. 3.—Intramural leiomyoma of posterior median wall of the lower third of the esophagus. Transpleural esophagotomy on the left side, with resection of tumor and reconstruction of wall of esophagus. *a* and *b*, moderate deformity of the lower part of the esophagus with some obstruction of the lumen shown by lateral roentgenogram on the patient's admission to the clinic.

sequent course was without incident, and at the time of his dismissal, two months after the operation, the cavity was healing well. The roentgenogram of the thorax revealed thickened pleura on the right and the roentgenogram of the esophagus was normal (fig. 2*b*).

CASE 2.—A woman, aged 58 years, consulted the clinic on April 21, 1947, because of attacks of belching of five years' duration. She stated that the belching would occur at any time and had no relation to food. She would have several periods of belching during the day but no pain. Eighteen months prior to her admission to the clinic, she had begun having a feeling of fulness in the lower part of the sternum, followed by a "grinding" sensation. On occasions she would have regurgitation of clear-colored frothy material. She also complained of

choking spells, when food, solid or liquid, would stick in her throat. She would sip water until food would pass on. The symptoms were periodic, troubling her for several days and then she would be free of discomfort for several days. She had no real pain. She had lost 20 pounds (about 9.1 Kg.) during eight to ten months prior to her admission. She had recently consulted her home physician, who diagnosed the condition as a polyp in the esophagus.

On examination at the clinic, the roentgenologic study revealed an intramural tumor involving the posterior medial wall of the lower third of the esophagus, which was thought to be a leiomyoma (fig. 3). Esophagoscopy revealed that the posterior and left lateral walls of the esophagus protruded into the lumen of the esophagus about 2½ inches (about 6 cm.) above the cardia. There was no difficulty in causing the esophagoscope to enter the stomach.

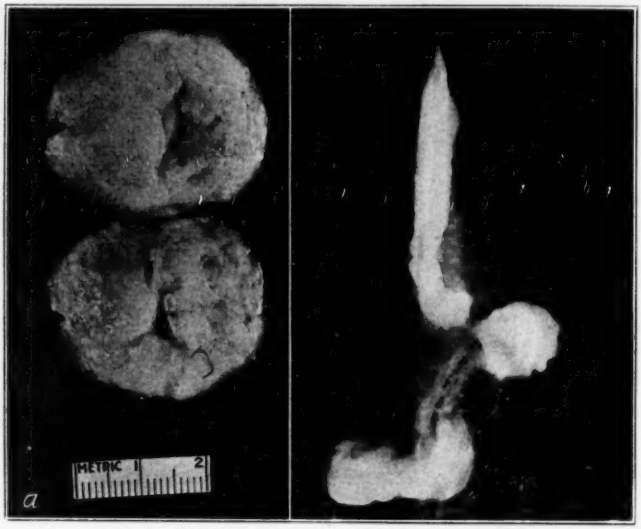



Fig. 4 (same case as in figure 3).—*a*, fairly well circumscribed leiomyoma measuring 3.5 by 3 cm. *b*, three weeks after operation. Moderate deformity at site of reconstruction of the wall of the lower part of the esophagus.

A diagnosis was made of an indeterminate lesion of the lower third of the esophagus, most likely a leiomyoma, and surgical intervention was advised.

On May 2, 1947, transpleural esophagotomy with complete resection of the tumor and reconstruction of the wall of the esophagus was performed on the left side. A posterolateral incision was made, the posterior two thirds of the seventh rib resected and the angles of the eighth and sixth ribs removed. The lower part of the esophagus was exposed just above the diaphragm, and a single well circumscribed leiomyoma was found in the left lateral wall. The tumor did not involve the mucous membrane, and it was located intramurally. It was carefully dissected from the wall of the esophagus, which was then reconstructed with

interrupted silk and chromic surgical gut sutures. The mediastinal pleura was sutured over the repaired esophagus. Temporary intercostal closed drainage was instituted.

The tumor measured 3.5 by 3 by 2.5 cm. (fig. 4 *a*). On microscopic study it proved to be a leiomyoma.

The patient's postoperative course was entirely uneventful, and she was dismissed from our care on the twenty-sixth postoperative day. The roentgenogram of the thorax revealed slight thickening of the pleura at the left base, and the roentgenogram of the esophagus was normal except for slight irregularity at the site of the operation (fig. 4 *b*).

CASE 3.—A man, aged 53 years, consulted the clinic on June 7, 1948. He gave a history of having had a persistent cough, for which he had consulted his home physician nine years prior to his admission to the clinic. A roentgenographic study had revealed a tumor in the lower third of the esophagus. Subsequent roent-

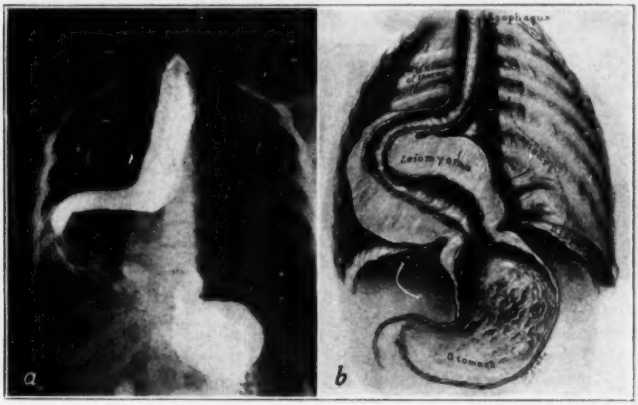



Fig. 5.—*a*, diffuse leiomyoma of the lower third of the esophagus and the cardial fourth of the stomach. Transpleural and transdiaphragmatic resection of the lower half of the esophagus and the cardial third of the stomach, with esophagogastroanastomosis, was performed on the right side. Roentgenogram on admission shows marked displacement of the lower half of the esophagus into the right thoracic cavity and questionable esophageal hiatal hernia. *b*, at operation, showing position of the diffuse leiomyoma of the entire muscular wall of the lower third of the esophagus, extending into the outer wall of the cardial fourth of the stomach.

genograms in 1941, 1943 and 1946 revealed no change in the size of the tumor. A recheck in 1948 revealed that it had increased from 4 cm. in diameter to about 10 cm., with pressure on the esophagus and displacement of it. The opinion was that it was either a posterior mediastinal tumor displacing the esophagus or an intramural tumor of the esophagus. The patient had not experienced loss of weight, and the only symptom was sharp sudden pain in the lower part of the thorax, on the right side of the spinal column, of a few seconds' duration.

On examination at the clinic the roentgenogram of the thorax revealed a rounded mass in the right posterior-inferior mediastinum. The roentgenogram of the esophagus revealed a tumor in the right lower quadrant of the thorax attached

to the esophagus and distorting its lower end, retracting it somewhat to the right. It was thought to be an intramural tumor or a small esophageal hiatal hernia. Esophagoscopy revealed that the esophagus was obstructed by an extraesophageal mass. It was otherwise normal.

A diagnosis was made of a posterior mediastinal tumor extending into the right lower quadrant of the thoracic cavity and distorting the lower part of the esophagus or a leiomyoma of the lower part of the esophagus, and exploration was advised.

On June 28, 1948, transpleural esophagotomy with complete resection of the tumor and reconstruction of the esophagus was performed on the right side. A posterolateral incision was made on the right, with resection of the seventh rib and the angles of the sixth and eighth ribs. The pleural cavity was entered through the posterior periosteum of the seventh rib. On exploration of the lower part of the thorax, a large tumor was found involving the lower 5 inches (about 13 cm.) of the esophagus and infiltrating two thirds of the right wall of the esophagus. The tumor, which was multicentric, was enucleated from the wall of the esophagus.

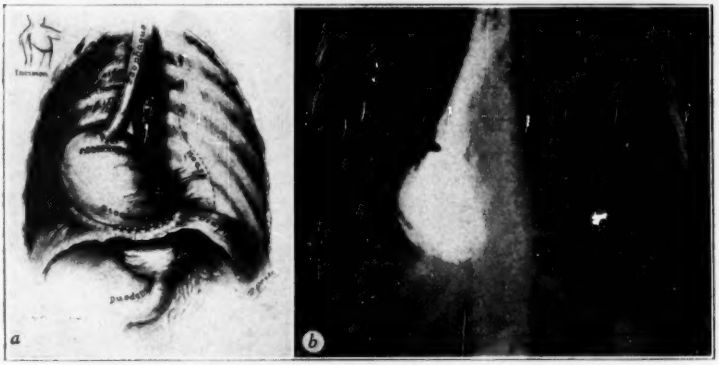


Fig. 6 (same case as in figure 5).—*a*, technic of transpleural esophagogastroanastomosis on the right; *b*, one month after operation. Half of stomach above the diaphragm. Esophagogastroanastomosis free.

There was considerable fixation of the mucous membrane in the center of the tumor, and it was necessary to resect about 3 cm. of the mucous membrane with the tumor. The mucous membrane was then closed with inverting sutures of silk and continuous surgical gut. The wall of the esophagus was then reconstructed, and the mediastinal pleura was sutured over the closure. Transcostal closed drainage was established after resection of a segment of the ninth rib.

The tumor measured 10 by 7.5 by 5.5 cm. On microscopic study it proved to be a leiomyoma.

The patient's postoperative course was without incident. He was dismissed from the clinic on the twenty-seventh postoperative day. The roentgenogram of the esophagus revealed postoperative changes in the lower end and a small esophageal hiatal hernia.

Case 4.—A woman, aged 19 years, consulted the clinic on May 6, 1947. She complained of choking spells of three years' duration. In April 1944 she had had a burning sensation in the epigastric region and also a choking sensation accom-

panied with a feeling of not being able to swallow or to get sufficient air into the lungs. Her symptoms progressed for two years. A roentgenogram was then taken by her home physician, who made a diagnosis of diaphragmatic hernia and referred the patient to the clinic. At the time of her admission she had spells of choking on solid food when eating rapidly but not when taking liquids. She had no pain but a burning sensation in the substernal region.

On examination at the clinic the roentgenogram of the chest revealed a mass in the lower lobe of the right lung near the midline, extending laterally just above the diaphragm and containing a segment of the alimentary tract. The roentgenogram of the esophagus revealed marked displacement of the lower half to the right (fig. 5 *a*), which was thought to be due to extrinsic pressure. The opinion of the roentgenologist was that the lesion was either a large mediastinal tumor or a hiatal diaphragmatic hernia. Esophagoscopy revealed considerable angulation of the esophagus to the right at the junction of the middle and upper thirds. There seemed to be a firm mass underlying the esophagus. It was impossible to determine whether it was an intrathoracic tumor extrinsic to the esophagus and displacing it or a tumor of the wall of the esophagus, as the esophagoscope could not be introduced beyond the middle third because of the angulation.

Since it was impossible to make a definite preoperative diagnosis, transthoracic exploration was advised. On June 20, 1947, transthoracic and transdiaphragmatic exploration was carried out on the right side, with resection of the lower half of the esophagus and the cardial third of the stomach, followed by esophagogastroanastomosis. A posterolateral incision was made in the right lower quadrant of the thorax, with resection of the eighth rib and of segments of the seventh and ninth ribs. On exploration of the thoracic cavity, a large tumor was found displacing and partially obstructing the esophagus. It involved at least the lower third of the esophagus and the cardial fourth of the stomach (fig. 5 *b*). The tumor involved the entire circumference of the esophagus as well as the entire circumference of the cardia and extended along the lesser curvature of the stomach. It was necessary to resect the lower half of the esophagus and the cardial third of the stomach. This resection was followed by an esophagogastroanastomosis (fig. 6 *a*). The esophagus was markedly thick walled, making the anastomosis difficult, but a satisfactory one was accomplished. The tumor measured 8 by 5 by 3 cm. On microscopic examination it proved to be a leiomyoma of a diffuse type which may be termed a leimyomatosis of the entire muscle wall of the esophagus. It did not involve the mucous membrane.

The patient's immediate convalescence was complicated by an irregular pulse and tachycardia, which improved after the fourth postoperative day, and her further course was satisfactory. She was dismissed on the twenty-fourth postoperative day. The roentgenogram of the esophagus revealed resection of the lower half of the esophagus and of one half of the remaining portion of the stomach above the diaphragm. The esophagogastroanastomosis was functioning freely (fig. 6 *b*). She returned one year after the operation, at which time her condition was satisfactory.

CASE 5.—A woman, aged 60 years, consulted the clinic on May 24, 1945, complaining of dysphagia and substernal pain of one year's duration. She stated that for the past year she had had increasing dysphagia associated with substernal pain, both of which had become severe. For six months she had had regurgitation of food while eating. This was occasionally associated with hemoptysis. For the past few months she had had some hoarseness. She also had had considerable dyspnea on physical effort, with some edema of her legs for several years.

On examination at the clinic, a roentgenogram of the esophagus and stomach revealed a tumor involving the distal third of the esophagus and extending to the cardia, displacing the esophagus to the left and anteriorly (fig. 7 *a*). The esophagoscopic examination showed an ulcerating polypoid lesion, which was extremely friable and necrotic, partially obstructing the lower third of the esophagus. Tissue was removed for biopsy and was reported as a malignant lesion, grade 4 (Broders' method), probably sarcoma.

Although the patient was a poor operative risk because of hypertension with cardiac damage and obesity, it was felt that operation was justified because of the impending complete obstruction.

On June 16, 1945, transthoracic and transdiaphragmatic resection of the lower half of the esophagus and the cardial end of the stomach and esophagogastroanastomosis were performed. A posterior lateral incision was made on the left,

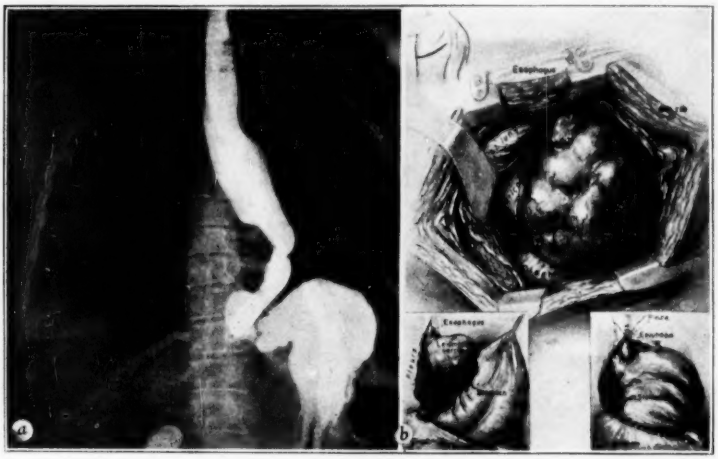



Fig. 7.—Diffuse leiomyosarcoma involving the lower third of the esophagus. Transpleural and transdiaphragmatic resection of the lower half of the esophagus and the cardial end of the stomach, with esophagogastroanastomosis, on the left side. *a*, tumor of the lower third of the esophagus extending to the cardia, displacing the esophagus to the left, with partial obstruction of the esophagus; *b*, at operation, showing diffuse leiomyosarcoma involving the lower third of the esophagus and technic of esophagogastroanastomosis.

with resection of the posterior two thirds of the ninth rib. The pleural cavity was opened through the posterior periosteum of the rib. The lower part of the esophagus was exposed by incising the mediastinal pleura, which revealed a large oval tumor involving the lower third (fig. 7 *b*). The tumor had the gross appearance of a multicentric diffuse type of leiomyoma. The esophagus above the tumor was separated from the surrounding mediastinal structures, and a piece of tape was placed around it above the tumor. The left phrenic nerve was cut, relaxing the diaphragmatic muscle. An incision was made through the anterolateral portion of the diaphragm extending through the esophageal hiatus. The stomach was then mobilized and the cardial 3 inches (about 8 cm.) was resected with the lower third

of the esophagus which was involved by the tumor. An esophagogastroanastomosis was made with the interrupted silk sutures and continuous chromic surgical gut. It was necessary to bring about two thirds of the stomach above the diaphragm in order to make the anastomosis without tension on the suture line. The opening

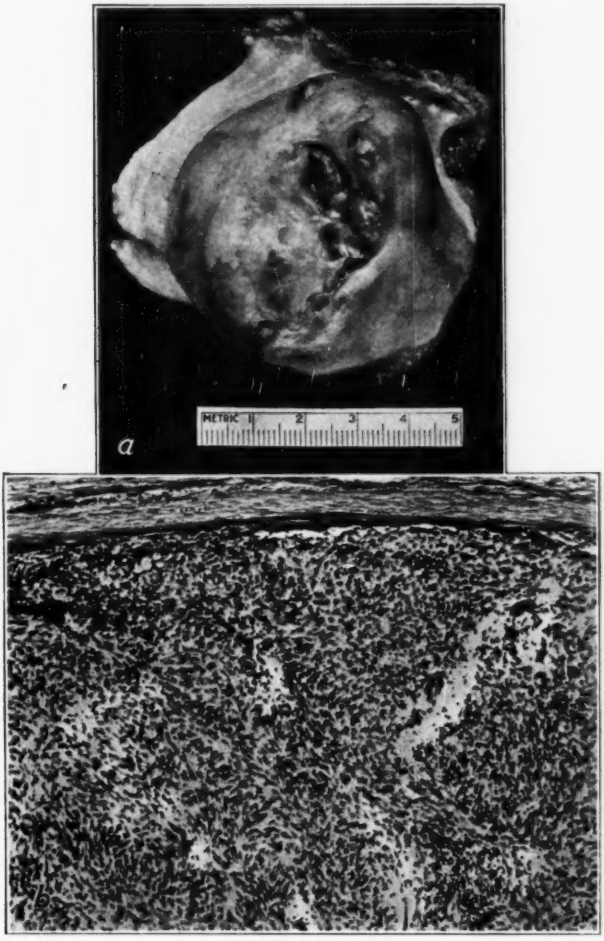



Fig. 8 (same case as in figure 7).—*a*, diffuse leiomyosarcoma; *b*, leiomyosarcoma, grade 4; × 75.

in the diaphragm was then partially closed, and the remaining opening was sutured to the walls of the stomach. The thoracic wall was closed with transcostal closed drainage through the posterior periosteum of the ninth rib.

The tumor measured 6 by 5.5 by 4.5 cm. (fig. 8 *a*). The microscopic study revealed a leiomyosarcoma, grade 4, invading and ulcerating the mucous membrane (fig. 8 *b*).

The patient's convalescence was satisfactory. The tube was removed from the pleural cavity on the fifth day. On the seventh day the temperature was elevated to 100 F. A roentgenogram revealed some signs of fluid in the left pleural cavity, and pleurocentesis was done, with removal of 200 cc. of clear fluid, which was sterile on culture. The temperature returned to normal. The patient was placed on a soft diet at the end of one week. She was dismissed six weeks after the operation, at which time she was taking a normal diet without difficulty. The roentgenogram revealed that the anastomosis between the esophagus and the stomach was entirely free and approximately two thirds of the stomach was above the diaphragm (fig. 9).

CASE 6.—A woman, aged 60 years, consulted the clinic on Sept. 23, 1948, complaining of difficulty in swallowing of three weeks' duration. She stated that

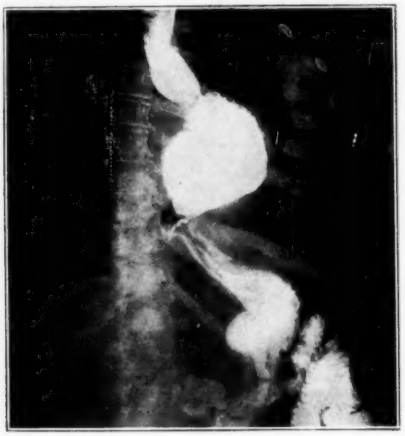


Fig. 9 (same case as in figure 7).—Roentgenogram made on dismissal of the patient six weeks after operation. Resection of the lower third of the esophagus was carried out. Two thirds of the stomach is above diaphragm. Esophagogastroanastomosis free.

she had had a sensation of solid food sticking in her throat for the past week. She gave little attention to this but did mention it to her physician while being attended for eye refraction. She had had no vomiting but had regurgitated some mucus. She had had no pain. Only liquids and soft food had been taken for the previous two weeks. These were ingested with ease as long as they were taken slowly. A roentgenogram made by her home physician revealed an obstructiong lesion of the lower end of the esophagus.

On examination at the clinic a roentgenogram of the thorax revealed a fusiform shadow overlying the mediastinum and extending below the diaphragm. This was thought to be a dilatation of the lower part of the esophagus or possibly a carcinoma of the cardia with esophageal obstruction. The roentgenogram of the esophagus revealed a carcinoma involving the lower third and the cardial end of the stomach.

Esophagoscopy revealed a large polypoid tumor mass in the lower part of the esophagus. A biopsy revealed a leiomyosarcoma, grade 3.

A left transpleural exploration was advised. The procedure was carried out on October 1. A posterolateral incision was made, with resection of the posterior three fourths of the seventh rib and removal of the angles of the sixth and eighth ribs. The pleural cavity was opened through the posterior periosteum of the seventh rib. There was a small amount of fluid in the left pleural cavity. On exploration of the lower part of the esophagus, it was found that there was a large edematous tumor involving the lower half of the esophagus. There was pronounced edema in all the surrounding tissues, with infiltration of the tumor into the pericardium and into the wall of the descending aorta. The upper portion of the tumor had infiltrated the wall of the pulmonary vessels and the bronchus of the left lung. There was notable fixation to the diaphragm. Tissue removed from the region of the extension proximal to the wall of the aorta was reported as leiomyosarcoma, grade 3. Because of the extensive involvement of the esophagus, with invasion into the surrounding structures, the condition was inoperable, and the chest was closed, with transcostal closed drainage established through the posterior periosteum of the seventh rib.

The patient's postoperative course was satisfactory. Some difficulty in swallowing solid food developed, and the esophagus was dilated. She returned home on the twenty-fourth postoperative day. She was placed on a bland diet, which she could take without difficulty. However, she was advised that further dilations would probably be necessary subsequently.

### COMBINED DATA ON ELEVEN CASES

The following is a summary of the 11 cases of tumor of the esophagus in which I have operated, 5 of which were previously reported and the remaining 6 reported in the present paper.

Among the entire series of 11 cases, in 2 the tumors were submucosal pedunculated benign tumors extending into the lumen of the esophagus. One of these was a lipoma covered with normal mucous membrane originating from a single pedicle at the introitus and extending to the cardia of the stomach; the other consisted of two large fibromyomas covered with normal mucous membrane, and each had a separate long narrow pedicle which originated from a single pedicle at the introitus of the esophagus.

Nine of the tumors were intramural tumors originating from the muscle of the wall of the esophagus. Seven of these growths were benign leiomyomas and 2 (18 per cent of the total 11 cases) were malignant leiomyosarcomas. The 7 benign leiomyomas involved only the muscular coat of the esophagus and did not involve the mucous membrane. Five were in the lower third and 2 were in the middle third of the esophagus. They were of three types according to gross characteristics. One was a single, completely encapsulated, circumscribed growth, involving a relatively small segment of the outer esophageal wall; 4 were multiple leiomyomas of multicentric origin involving a large segment and from one third to two thirds of the circumference of the wall of the esophagus;

the remaining 2 were a diffuse type of multiple leiomyoma, which may be termed "leiomyomatosis," involving the entire circumference of the outer wall of the lower part of the esophagus and for a longitudinal distance of from 3 inches (about 8 cm.) to one third of the entire length of the esophagus. One of these growths extended into the cardia of the stomach.

The 2 malignant growths or leiomyosarcoma were of the diffuse type involving the entire circumference of the lower 3 to 5 inches (about 8 to 13 cm.) of the esophagus and also invaded the mucous membrane, producing areas of necrosis and ulceration.

Of the entire series of 11 patients, 6 were women and 5 were men. The youngest was 19 and the oldest 60 years of age. The average age was 44 years.

The duration of symptoms was one to fifteen years. Five of the 11 patients presented symptoms of esophageal obstruction. Of these, 2 had an intramural pedunculated benign growth, 2 a malignant growth and the remaining patient a benign intramural tumor markedly distorting and involving the lower third of the esophagus as well as the cardial fourth of the stomach.

In only 1 case was there any hemorrhage from the lesion, and in that case the lesion was malignant. In 2 cases the tumor appeared in the mouth during forceful vomiting. In each of these cases the tumor was a pedunculated benign submucosal growth.

The surgical procedures carried out in the 11 cases of this series were as follows: The 2 submucosal pedunculated tumors were completely removed after the pedicle had been cut and ligated at their origin after performance of esophagotomy, 1 through a left cervical approach and 1 through a right posterolateral transpleural approach. The 7 benign intramural leiomyomas were completely removed, 5 by excision of the tumor from the muscular wall of the esophagus and reconstruction of the remaining portion of the wall of the esophagus. In 3 of these 5 cases the tumor was excised extramucosally, and in 2 the mucosa was extremely thin and was incised and closed, with the reconstruction of the outer wall of the esophagus. The approach for these 5 tumors was right transpleural in 3 cases, left transpleural in 1 case and left abdominal in 1 case. In the remaining 2 cases of benign lesions the tumor involved the entire wall of the lower part of the esophagus, 1 involving the cardia of the stomach also. These 2 tumors were completely removed by transpleural and transdiaphragmatic resection of the lower part of the esophagus and the cardial end of the stomach, with esophagogastroanastomosis. The approach for 1 was through the right thoracic cavity and for the other through the left thoracic cavity.

The 2 malignant leiomyosarcomas were operated on through a left posterolateral transthoracic approach. In 1 case the tumor was found to

be inoperable because of the extension of the malignant lesion into the mediastinal structures and the wall of the aorta. In the remaining case the tumor was completely removed by transthoracic and transdiaphragmatic resection of the lower part of the esophagus and the cardia of the stomach followed by esophagogastroanastomosis.

## SUMMARY

In 1 of 2 cases of malignant tumors, the lesion was inoperable and the patient continues to have trouble but is being partially relieved by esophageal dilations. The other patient with a malignant lesion was completely relieved after operation and has progressed satisfactorily without further trouble during three years after operation.

The results obtained in the 9 cases in which the tumor was benign are as follows: In 1 case bilateral pneumonia developed postoperatively and the patient died on the fourth day. In 2 cases localized empyema developed postoperatively. Drainage of the empyema was followed by complete recovery. Six patients had no postoperative complications and made complete recoveries from the operation.

One patient died, in 1 case the lesion was inoperable and 9 patients recovered from operation and have been relieved of symptoms.

# SURGICAL TREATMENT OF CARCINOMA OF THE ESOPHAGUS AND STOMACH

HAROLD LINCOLN THOMPSON, M.D., Ph.D.
LOS ANGELES

WHILE surgery has made extraordinary advances on several fronts during the last decade, one of the most significant is the transthoracic attack on lesions of the esophagus and gastric cardia. Thus by the expedient of a new approach, one of the greatest needs in surgical treatment of malignant disease has been fulfilled.

In order to appreciate this, it is only necessary to refer to the vital statistics of the United States.[1] In the year 1945 there were 3,100 deaths from cancer of the esophagus while deaths from carcinoma of the stomach numbered 25,832. If it was true then as now that an estimated 10 per cent of all cancers of the stomach occurred in or involved the cardia, there were 2,583 cases of carcinoma of this region. Thus, if to the 3,100 carcinomas of the esophagus are added 2,583 carcinomas of the cardia, there were in 1945 a total of 5,683 cases in which carcinoma occurred at the esophageal and cardiac levels. This total represents the number which had they occurred seven years previously would have been classified by roentgenologists as "inoperable" according to the practice of that period. Whereas gastric cancer now heads the list in frequency, cancer of the esophagus continues to be more common than cancer of the lip, tongue, mouth, pharynx, larynx, thyroid or kidney or all malignant tumors of bone. In view of these vital statistics, the significance of this most recent approach to surgical treatment of malignant disease of the upper part of the alimentary tract immediately is apparent.

## ANATOMY

Complete agreement among students of anatomy of the human stomach does not exist. Anatomic concepts vary with the point of view of the observer. Thus descriptive gross anatomy varies from histologic and physiologic anatomy. Clinically, there are the surgical, the roentgenologic and the gastroscopic concepts of anatomy, each having its legitimate place according to the circumstances in which

Read at the Fifty-Sixth Annual Meeting of the Western Surgical Association, St. Louis, Dec. 3, 1948.

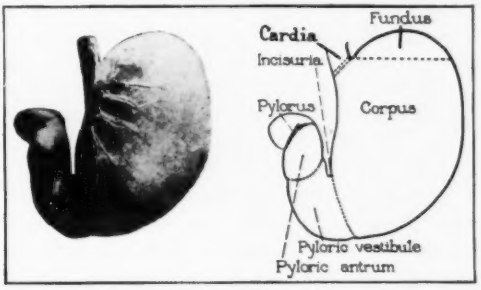
1. Vital Statistics of the United States, 1945, United States Public Health Service, National Office of Vital Statistics, 1947, pp. 1-10.

anatomic observations are made. It is not always possible, therefore, to reconcile divergent points of view into a single descriptive anatomy of the human stomach.

One of the greatest points of variance anatomically is with respect to the gastric cardia. Few anatomists state precisely what the limits of the cardia are. Thus in the same text one author refers to the cardia as the "region of the junction of the esophagus with the stomach" and illustrates it as that entire portion of the stomach proximal to the incisuria.

Previously I [2] have pointed out the following facts:

Histologic and physiologic investigations have demonstrated that the simple stomachs of mammals are divided into different parts with different functions and properties. There are two great divisions of the simple stomach, the plane of demarcation being indicated by the incisuria angularis (figure). These divisions




Drawing of the stomach indicating its subdivisions. The cardia is incompletely defined grossly but clearly delimited histologically as an annular zone approximately 5 mm. in width surrounding the esophageal orifice of the stomach.

are the proximal portion, named the fundus, and the distal portion, called the pylorus. The fundus is divided into the cardia, a narrow zone about the esophageal orifice, and the corpus or prepyloric portion occupying the region between the cardia and the incisuria. The pylorus is divided into three subdivisions; a proximal portion, the pyloric vestibule; an intermediate portion, the pyloric antrum; and a distal portion or pyloric canal formed by the pyloric sphincter. . . . The cardia is lined by columnar epithelium containing mucous glands which resemble those of the pylorus.

The cardia, therefore, while being grossly the general and incompletely defined region of the esophageal entrance is, on the other hand, clearly delimited on a histologic basis as an annular zone approximately 5 mm. in width surrounding the orifice of the esophagus. The glands of the cardia may be regarded as modified fundus tubules having cells which

2. Thompson, H. L.: Studies on the Surgery of the Stomach, Thesis, University of Minnesota, 1930, pp. 7-9.

resemble the chief cells of the gastric tubules along with a few parietal cells and in addition the distinctive tubules which recall the intestinal crypts of Lieberkühn.

From the foregoing it is clear that the mucous membrane of all portions of the stomach, including the cardia, is of the glandular type. In contradistinction to this, the mucous membrane of the contiguous esophagus is of the squamous epithelial variety. This important distinction between the histologic nature of the esophagus and that of the cardia has significant pathologic and, therefore, clinical implications.

## SURGICAL ANATOMY

Malignant disease existing in the gastric cardia or the distal esophagus presents the same surgical problem. The transthoracic approach now has made accessible to surgical attack these two previously almost inaccessible organs. Thus, while differing in structure and function, these viscera constitute a single surgical region. With the exception of Garlock,[3] most writers treat them as a single anatomic or therapeutic region. He is the only extensive writer who is consistent in his differentiation between lesions of the esophagus and those of the stomach. His distinction is based chiefly on the histopathologic nature of the respective malignant lesions.

While it cannot be denied that the pathologic process is a sound basis for such distinction, there are several reasons why consideration of the surgery of these two regions together should be continued: First, the esophagus and the cardia are contiguous anatomically. Second, the most frequent early symptom of a lesion existing at either site is dysphagia. Third, diagnostic confirmation usually is dependent on esophagoscopy with biopsy. Fourth, successful removal of a given malignant lesion is dependent on excision of a portion of the esophagus or the stomach or both. Thus, from an anatomic, symptomatic, diagnostic and therapeutic standpoint, the contiguous portions of the esophagus and stomach constitute a single surgical region.

Anatomically there are three types of lesion accessible by the transthoracic approach, namely, lesions of the esophagus and the cardia and lesions of the body or fundus of the stomach which extend to the cardia.

## PATHOLOGIC PROCESS

It has been stated that Garlock [3] reported his surgical procedures on the esophagus and gastric cardia on a histopathologic basis. Since the mucosa of the esophagus is squamous in type, carcinoma originating in this organ is of the corresponding squamous cell variety. Since, on

3. Garlock, J. H.: The Problem of Cancer of the Esophagus, J. Mt. Sinai Hosp. **7**:349-352, 1940.

the contrary, the mucosa of the cardia is glandular, carcinoma of this region is adenocarcinomatous in type. Aside from the histologic distinction between the two types of carcinoma, the most characteristic difference is in the mode of spread. Adenocarcinoma of the stomach tends to spread to the esophagus directly and through the submucosal layer. Less frequently squamous cell carcinoma of the esophagus spreads to the stomach by direct invasion on the one hand and to the paracardial lymph nodes on the other. Perhaps the only exception which can be taken to these pathologic distinctions is the theoretic possibility of the origin of adenocarcinoma from rests of gastric mucosa situated in the esophagus.

## NOMENCLATURE OF OPERATIVE PROCEDURES

In view of these anatomic and pathologic considerations, I believe that, in the reporting of additional cases, attention should be given to the nomenclature of the operative procedures employed. Since the distal portion of the esophagus and the proximal end of the stomach constitute one surgical region, there is no reason to abandon the present practice of reporting cases of carcinoma of the respective areas together in the surgical literature. In order, at the same time, to distinguish lesions arising in the esophagus from those originating in the stomach, irrespective of extension from one organ to the other, I should like to suggest the employment of two new terms. I would suggest the term esophagocardiectomy for surgical removal of lesions primary in the esophagus and cardioesophagectomy for lesions originating in the cardia. This distinction is made in the series of cases reported here. In the future, as the number of cases in the literature increases, refinement in nomenclature should become increasingly important.

## HISTORY

The history of esophagectomy and cardiectomy appears to be of interest. I have divided the history into four periods accordingly as each period is characterized by a significant development in this field of surgery.

The first period dates from 1871 to 1906: In 1871 Billroth [4] performed esophagectomy in animals and thereby proved that the procedure was feasible. This was ten years before his pupil, Wölfler,[5] performed the first successful operation (gastrojejunostomy) on the human stomach. It is of interest also to note that in 1877 Czerny,[6]

4. Billroth, T.: Ueber de Resection des Oesophagus, Arch. f. klin. Chir. **8**:65, 1871.

5. Wölfler, A.: Gastroenterostomie, Zentralbl. f. Chir. **8**:705-708, 1881.

6. Czerny: Neue Operationen, Centralbl. f. Chir. **4**:433, 1877.

also Billroth's pupil, performed the first cervical esophagectomy. The patient survived. In 1895 Biondi [6a] performed transthoracic esophagogastrostomy on dogs. He apparently was the first to advance the stomach into the thorax. In 1898 Mikulicz [7] reported having performed transabdominal cardiectomy on a human being, but the patient did not survive. In 1904 Mikulicz,[8] using the Sauerbruch positive pressure chamber, performed transthoracic cardiectomy on a dog. The animal survived the operation for four weeks but died of inanition from implied stenosis at the anastomosis but without evidence of intrathoracic complication. The author pointed out that such a complication in man doubtless could be treated successfully by bougienage. More important than this, however, was the fact that the author, using the same technic on another animal, demonstrated the practical possibility of advancing the stomach into the thorax. At the time of his report, the animal was living six weeks after operation and appeared to be in excellent health. Wendel [8a] in 1906 performed transthoracic cardiectomy with esophagogastrostomy by use of the Murphy button. He reported his case as the first technically successful transpleural esophagogastrostomy.

The second period begins in 1908 and extends over the decade to 1918. This period is marked by the successful performance of cardiectomy and esophagectomy in human beings, with survival of the patients. In 1908 Voelcker [9] of Heidelberg performed successful transabdominal cardiectomy on a 64 year old woman. In 1912 Torek [10] of New York performed the first successful transthoracic esophagectomy. The patient survived twelve years after resection and died of pneumonia at the age of 80 years without demonstrable recurrence of the malignant lesion at autopsy. In 1916 and 1918, respectively, Brun [11] and Bircher [12] reported survivals following transabdominal cardiectomy.

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6a. Biondi, A.: Suppl. al Policlin. **1**:964, 1895.

7. Mikulicz, J.: Beiträge zur Technik der Operation des Magencarcinoms, Arch. f. klin. Chir. **57**:524-532, 1898.

8. Mikulicz, J.: Ueber Operationen in der Brusthöhle mit Helfe der sauerbruchschen Kammer, Deutsche med. Wchnschr. **30**:530-532 and 577-579, 1904.

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11. Brun, H.: Ueber die Bedeutung der Unterbindung der Arteria coronaria sinistra bei Resektionen des Magens insbesondere der Kardia, Deutsche Ztschr. f. Chir. **135**:81-100, 1916.

12. Bircher, E.: Operative Heilung eines Carcinoms am Übergang des Oesophagus in der Cardia, Cor.-Bl. f. Schweiz., Aertze **48**:467-478, 1918.

The third period in the history of these procedures extends for a brief five years, from 1933 to 1938. In 1933 Ohsawa [13] reported 2 cases of abdominothoracic esophagectomy, in 1 of which the patient survived. In 1937 Marshall [14] performed the first recent transthoracic cardioesophagectomy, with survival. In the following year Cattell [15] in 1 case and Ochsner and DeBakey [16] in another case performed successful cardioesophagectomy. In 1938 Adams and Phemister [17] performed the first single stage transthoracic esophagocardiectomy, with survival. At the time of this writing their patient is living,[18] ten years and ten months after operation. This work thus is credited with having inspired the modern endeavors in this difficult field.

The fourth and recent period extends from 1939 to 1947. On April 27, 1939, Garlock [19] performed his first single stage esophagogastrostomy, with survival. Garlock deserves great credit for having perfected and modified, prior to that report, the Torek technic of operation whereby in 1938 he was able to report 3 successful cases.[20] By 1940 [19] he had to his credit six successful operations by the modified Torek technic and one survival by the method under present consideration. In 1939 Churchill and Sweet [21] began their work, which has continued uninterruptedly to date. Sweet's contributions to the technical phases of the transthoracic approach have been outstanding. In addition he has the largest series of cases reported. In my opinion, this period closed in 1947, when Sweet [22] stated that the phase of technical development had ended and that the period of statistical evaluation of the various operative procedures had begun.

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13. Ohsawa, T.: The Surgery of the Esophagus, Arch. f. jap. Chir. **10**:605, 1933.

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15. Cattell, R. B.: Resection of the Lower Esophagus for Carcinoma, S. Clin. North America **21**:649-655, 1941.

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18. Phemister, D. B.: Personal communication to the author.

19. Garlock, J. H.: The Surgical Treatment of Carcinoma of the Thoracic Esophagus, Surg., Gynec. & Obst. **70**:566-569, 1940.

20. Garlock, J. H.: The Surgical Treatment of Carcinoma of the Thoracic Esophagus, Surg., Gynec. & Obst. **66**:534-548, 1938.

21. Churchill, E. D., and Sweet, R. H.: Transthoracic Resection of Tumors of the Stomach and Esophagus, Ann. Surg. **115**:897-920, 1942; **116**:566-573, 1942.

22. Sweet, R. H.: Carcinoma of the Esophagus and Cardiac End of the Stomach, J. A. M. A. **135**:485-490 (Oct. 25) 1947.

### CLINICAL FEATURES

At onset, the symptoms from carcinoma of the esophagus and cardia, while characteristic, usually are so mild that the patient frequently assumes erroneously that they originate from benign disturbance of the gastrointestinal, circulatory or even respiratory tracts. The earliest and usually the most persistent symptom is dysphagia. In the beginning it usually is mild, transitory or recurring. It arises from obstruction to the passage of food through the esophagus and into the stomach. Obstruction at these higher levels in the gastrointestinal tract is relievable by regurgitation and thus never amounts to the acute forms of obstruction such as occur beyond the pylorus. Neither is it associated with the marked dilatation and elongation of the esophagus seen in the chronic form of obstruction associated with cardiospasm. Ultimately the dysphagia progresses until it becomes distressing to the patient. His first visit to the physician often is deferred until this time.

While pain is not usual, it is, paradoxically, not an uncommon symptom. Usually it appears late and probably results from extension of or infection in the neoplastic growth. Occasionally bleeding, as manifested by hematemesis or melena, is the first symptom noted. If the patient presents himself early, the physician, therefore, must be alert to the possibilities of the mild symptoms.

In the later stages, while there may be pain or chronic anemia, the most characteristic feature is progressive loss of weight. At this stage and despite the lowering of general morale induced by starvation, the patient usually is eager to assume the risk of an extensive surgical procedure in order to obtain relief.

### DIAGNOSIS

The diagnosis of carcinoma of the esophagus or cardia usually is based on a history of persistent dysphagia combined with characteristic roentgenologic and endoscopic findings. In my experience pain has been a significant symptom in fewer than half the cases. Except for evidences of loss of weight or bleeding, the physical findings commonly are insignificant. Fluoroscopy is of the utmost value in detecting the presence of a lesion of the esophagus or gastric cardia. Occasionally, however, especially if situated at the cardia, the lesion escapes detection by fluoroscopy, whereon diagnostic esophagoscopy or gastroscopy must be relied on. In view of the fact that relief in these cases requires extensive surgical procedure, it is not only desirable but obligatory to confirm the roentgenologic findings by endoscopy. Biopsy furnishes valuable and final proof of the existence of a malignant process.

### TRANSTHORACIC AND ABDOMINOTHORACIC APPROACHES

During the last half decade, the transthoracic approach has become established in gastric and esophageal surgery. Thus many patients who, prior to a decade ago, were denied the benefits of surgical relief, if they were living now might find their condition amenable to it. Moreover, this approach has permitted improvement in the technic of certain other procedures as, for example, total gastrectomy and the surgical treatment of cardiospasm and stricture of the esophagus.

A logical extension of the transthoracic route is the abdominothoracic approach to lesions primary in the corpus, cardia, or terminal part of the esophagus or where there is possibility of extensive metastasis to the upper part of the abdomen. In fact, one of the first successful operations of the recent period, performed by Ohsawa [18] in 1933, was approached abdominothoracically. It was his report that led Phemister [18] and his associates to attack the problem of esophageal surgery, which resulted in their performance of the first successful single stage esophagogastrectomy with the result that the patient is living now, ten years and ten months after operation. Since that time the value of the combined approach has been emphasized by Garlock.[23] He enumerated several advantages: (1) that operability may be determined through a small upper left rectus incision before making the incision into the thorax; (2) that in no other way, prior to opening the thorax, may the surgeon determine the presence or absence of hepatic metastasis, fixation of the growth, the presence of peritoneal implants on the diaphragm or in the pelvic peritoneum or extensive retroperitoneal involvement as in the all-important area about the celiac axis.

The success of these approaches to the surgery of the alimentary tract is the result of important technical advances in anesthesiology, thoracic surgery, the care of the depleted patient, the application of antibiotic therapy and chemotherapy and the successful handling of pulmonary and vascular complications. The wide exposure of the esophagus and proximal portion of the stomach makes these operations ideal for resection of lesions involving the esophagus, the cardia, the body and the fundus of the stomach. Without question, they are the best approaches for total gastrectomy because of the excellent exposure they provide for the removal of involved lymph nodes and contiguous structures and for esophagojejunal anastomosis. The advantages with respect to lesions in the stomach are, first, that they permit total gastrectomy if needed and, second, that they permit resection of the proximal portion of the stomach, with preservation of the distal portion in cases in which total gastrectomy otherwise might be required. With

23. Garlock, J. H.: Combined Abdominothoracic Approach for Carcinoma of Cardia and Lower Esophagus, Surg., Gynec. & Obst. **83**:737-741, 1946.

respect to carcinoma of the esophagus, they replace the Torek operation, which no longer is adequate and thus is obsolete.

### PREPARATION OF THE PATIENT

In the early cases, loss of weight and anemia usually are insignificant and thus require little attention during the period of preparation of the patient for operation. In late cases, however, malnutrition may be severe, and steps must be taken to correct it. Dehydration and hypoproteinemia may be correctable by the intravenous administration of dextrose, electrolytes and amino acids. Occasionally, however, the prolonged delay required to correct severe hypoproteinemia is not warranted, and one must proceed with surgical intervention before the condition is overcome entirely. In patients in whom anemia is a problem, infusion of red blood cell concentrates or of whole blood may be required. If obstruction is complete, it may be necessary to wash out the esophagus daily. Appropriate doses of liver extract and vitamins B and C should be given for several days prior to operation. For twenty-four hours before operation sulfadiazine and penicillin are administered, and 0.5 Gm. of streptomycin in solution is given by mouth. A Levin tube is inserted on the morning of operation, and the entire thorax, axilla and abdomen must be shaved so that, if necessary, the incision may be extended upward or downward.

### ANESTHESIA

A competent anesthetist is indispensable to the successful application of this type of surgical procedure. Not only must the anesthesia be adequate but the condition of the patient must be entrusted to his care for a period of four or five hours. Not only must anesthesia be maintained but the patient's circulatory and respiratory systems must be kept under constant observation. The anesthetist must be adept at intratracheal technic, and he must understand the pharmacology of the newer anesthetic agents and adjuvants. Replacement of fluids by parenteral administration or by the transfusion of 500 to 1,500 cc. of blood usually is necessary to insure adequate blood pressure levels during the course of the operation. If cardiac arrhythmias appear as a result of vagal stimulation, the anesthetist must report it so that perineural infiltration of procaine hydrochloride may be administered by the surgeon. The addition of 1 or 2 Gm. of procaine hydrochloride to the crystalloid solution given intravenously may prevent arrhythmia. If severe arrhythmia supervenes, the anesthetist may give 50 to 100 mg. of procaine intravenously in a single dose. The compressed lung must be inflated at thirty or forty minute intervals during the operation. Tracheobronchial aspiration is required occasionally during and always after operation to avoid atelectasis. Tension on the diaphragm may be

controlled by the competent administration of curare. Positive intrathoracic pressure must be applied at the conclusion of operation for the dual purposes of complete aeration of the compressed lung and expulsion of the exudate which has accumulated in the thorax during the operation. It is my observation that, with the exception of the surgeon, the anesthetist is the busiest man on the job.

The article on anesthesia by Dr. Harry Brown, which follows this paper, may be consulted for greater detail.

## TECHNIC OF OPERATION

The history of the development of the technic of operation has been covered ably by Bird,[24] Ochsner and DeBakey[16] and Pack and McNeer.[25] It has been pointed out that Sweet and Garlock have contributed greatly to the standardization of technic.

While most writers stress the great advantage which has resulted from the transthoracic approach, I believe that there is another feature which is of equal importance and which has not received the attention in the literature that it deserves. That is the development of the technic of advancement of the stomach or jejunum into the thorax in order to complete esophagogastric or esophagojejunal continuity. In 1904 Mikulicz[8] operated on 2 dogs in the Sauerbruch positive pressure chamber. In 1 dog he advanced the stomach into the thorax, and in the other he performed transthoracic esophagectomy. At the time of his report the first dog was living and in good health six weeks after operation. The second dog, on the contrary, lived for four weeks and died of inanition from implied stenosis but without evidence of other complication. Cattell,[15] as recently as August 1938, utilized advancement or "reduction" of the stomach into the thorax transperitoneally as a stage preliminary to subsequent successful transthoracic cardioesophagectomy.

By means of the transthoracic and abdominothoracic approaches it now is possible to apply surgical treatment not only to carcinoma limited to the esophagus, cardia and the body of the stomach but also when invasion has extended to adjacent organs. In ideal cases they permit removal of the lesion, adjacent involved organs and regional lymph nodes, with prospects of cure. Essentially the operations consist either of thoracotomy or of abdominothoracotomy, with radical excision of a segment of the esophagus, the cardia, fundus or corpus of the stomach, or even total gastrectomy, with removal of the majority

24. Bird, C. E.: Recent Advances in Surgery of the Esophagus, Surgery **6**:617-637, 772-801 and 949-975, 1939.

25. Pack, G. T. and McNeer, G.: Surgical Treatment of Cancers of the Gastric Cardia, Surgery **23**:976-1019, 1948.

of regional lymph nodes, and esophagogastric or esophagojejunal anastomosis within the thorax or even within the base of the neck. Thus, the internal restoration of continuity of the digestive tract which these operations provide offers palliation superior to that of any procedures yet devised.

The technic of operation as utilized in the cases reported here is as follows: In the transthoracic approach the chest is opened between the ribs or, as is usual, through the beds of the seventh, eighth or ninth ribs. In cases of high esophageal lesions the seventh, sixth, fifth and even fourth ribs may have to be divided in order to reach the apex of the thorax. The thoracic cavity first is explored, and any pleural adhesions which may be present are divided with care. Since the advent of curare, it no longer is necessary to avulse, divide or crush the phrenic nerve to overcome resistance of the diaphragm. By its use relaxation of the abdominal muscles also may be secured. The mediastinum is divided posteriorly to the pulmonary ligament, and with blunt finger dissection the distal portion of the esophagus is explored, care being taken to note any apparent involvement of the mediastinal lymph nodes. The distal end of the esophagus then is mobilized over a Penrose drain.

An incision is made through the dome of the diaphragm, and the stomach, regional lymph nodes, liver and peritoneal surfaces are explored manually. At this point, should the lesion prove nonresectable, it only is necessary to close the diaphragm, the mediastinum and the thoracic cage to conclude the operation. If it is decided to proceed with resection, the diaphragmatic incision is extended into the esophageal hiatus, thus creating of the upper abdominal and left thoracic regions a single cavity. If the lesion is situated in the proximal portion of the stomach, resection of that portion of the stomach with the regional nodes is carried out, preferably without removal of the spleen. After the distal portion of the stomach is closed, the esophagus is divided, and an end of the esophagus to side of the stomach anastomosis is completed by the use of three layers of interrupted silk sutures. If the lesion is limited to the esophagus, that organ is freed from its surrounding structures, with due consideration of its blood supply. The stomach then is mobilized and advanced into the thorax to the degree that is required to make a suitable anastomosis either below or above the arch of the aorta. The stomach thus permanently becomes a thoracic organ either partially or wholly. It then is anchored to the mediastinal and parietal pleura, and the diaphragm is closed by suturing it loosely to the stomach walls so as not to produce constriction and thus induce the symptoms which are characteristic of esophageal hiatus hernia.

In case the performance of total gastrectomy is required, it is necessary and simpler to advance a loop of jejunum into the thorax for

esophagojejunal anastomosis. In these circumstances jejunojejunostomy between the proximal and distal loops seems to me to be not only desirable but obligatory.

If the lesion is situated primarily in the corpus of the stomach, with involvement of the cardia, and if there is extension to lymph nodes or other organs below the diaphragm, the combined abdominothoracic incision is extremely valuable. In this procedure an incision through the outer margin of the left rectus abdominus may be made. Through it operability is determined by systematic palpation of the stomach, liver and suprapyloric, supragastric, paracardial, pancreaticolienal, middle colic and infrapyloric lymph nodes. The presence or absence of hepatic nodules or of peritoneal implants under the diaphragm, in the region of the vasa brevia, about the middle colic artery, in the great omentum and in the cul-de-sac thus may be determined. If the tumor appears to be resectable, the incision in the skin is extended upward to the costal arch, where it is extended laterally between the eighth and ninth ribs as far as the medial border of the scapula. The cartilagenous costal arch between these ribs is divided, as are the muscles down to the pleura. Through an initial opening in the pleura a finger is inserted and used as a guide to avoid injury to the lung while the pleural incision is advanced. The diaphragm is incised from its peripheral rib attachment to the esophageal hiatus, with ligation of the phrenic vessels and the small but important vascular branches about the esophageal hiatus. From this point on, the procedure is carried out as described previously, with certain advantages: 1. All manipulations may be carried out under direct vision. 2. The left gastric artery is more easily ligated near its origin from the celiac axis. 3. The lymph nodes in the important area about the celiac axis may be excised more completely. 4. The surgeon may resect involved adjacent organs such as the spleen, the tail of the pancreas or the colon if necessary. 5. Should total gastrectomy be necessary, visualization of the jejunal blood supply is more satisfactory.

Closure of this incision is carried out in the usual manner except that the costal arch must be repaired by approximating the costal cartilages with absorbable or nonabsorbable suture material. The thoracic portion of the wound is closed in layers with interrupted silk. Closure of the abdominal incision is facilitated to a marked degree by first inserting through-and-through sutures of strong material such as steel wire or silk.

### POSTOPERATIVE CARE

The postoperative care of patients who have undergone surgical treatment for carcinoma of the esophagus or cardia is extremely impor-

tant. As pointed out by Sweet,[26] this is abdominal surgery with thoracic surgical postoperative care.

Before the patient leaves the operating room, the anesthetist should aspirate the tracheobronchial tree. He should ascertain that the blood pressure is at a satisfactory level and that the patient's color is good. If either of these is not satisfactory, intravenous administration of fluids or blood must be continued after the patient has returned to his room and oxygen must be administered intranasally for twenty-four hours or more until the color of the skin improves. Orders are left that the patient is to be turned from side to side at half-hourly intervals and that he is to take six or eight deep inspirations at hourly intervals. The administration of carbogene by mask is exceedingly helpful in securing the patient's cooperation with deep breathing. Coughing should be encouraged to the extent that less rather than more analgesic should be given. If too much analgesic is administered, the respiratory center may be depressed and the patient may not inspire as deeply as he should. On the other hand, if too little is given, he may fail to do so because of pain. There is no instance in which better judgment is required on the part of the nursing personnel than with respect to the administration of analgesics in the early period after operation. Experienced, special nursing is a distinct asset in the conduct of postoperative care in these cases. At present, I prefer to use demerol hydrochloride® in doses of 50 to 100 mg., depending on the weight, age and tolerance of the patient, administered at four to six hour intervals.

Suction on the intrathoracic Foley catheter is maintained for from two to three days, during which time 350 to 500 cc. of serosanguineous fluid usually is removed from the thoracic cavity. When the drainage ceases, the catheter is removed and the stab wound allowed to close spontaneously. In these circumstances it seldom is necessary subsequently to aspirate pleural effusion.

Nutrition is maintained for the first three days by the administration of 1,000 cc. of amigen® and 1,000 cc. of 5 to 10 per cent dextrose in sodium chloride solution. Sips of water are allowed from the outset while constant Levin suction is employed. On the third or fourth day the Levin tube is clamped off for four to six hours while the sips are continued. If they are tolerated by the patient, the Levin tube is removed at the end of the test period. Thereafter thin liquids are given for one day and soft foods for two or three days, after which a general diet is allowed. Ileus usually is minimal and, therefore, seldom is a problem when the thoracic approach is used. There is little gaseous distention or abdominal distress. This is in distinct contrast to what is

26. Sweet, R. H.: Personal communication to the author.

common experience after laparotomy. Nevertheless, it is the practice to administer neostigmine in dilution of 1 : 2,000 every six hours for the first seventy-two hours.

Close observation of the chest is mandatory for the first two to five days, during which auscultation should be carried out at six hour intervals. The appearance of pulmonary complications usually is indicated by an abrupt rise in pulse rate, which ordinarily precedes a rise in temperature. If, accompanying these, there are diminished breath sounds in any portion of the chest, beginning atelectasis is assumed. In such circumstances or in case of doubt, tracheobronchial aspiration is carried out with the use of the nasal catheter. This procedure is repeated as often as is necessary until aeration is adequate in all portions of the thorax.

The aid of the anesthetist is solicited during the early postoperative period. The anesthetist should be willing to become a clinician to the extent that he examine the patient's chart, palpate his abdomen and percuss and auscultate the chest. Thus, if the patient has the benefit of the combined attention of the surgeon, his assistant, the anesthetist and the special nurses, there is minimal chance that complications may reach the stage of irreversibility. It is much better to prevent complications than to treat them after they have developed. For this reason it is our practice to endeavor to anticipate complications and employ prophylactic rather than therapeutic measures.

If the functional capacity of the heart allows, the patient usually is assisted to stand at the side of the bed on the afternoon of operation and twice daily thereafter. Walking is encouraged as soon as the patient is able to do so. Usually the patient is able to leave the hospital on the seventh to ninth postoperative day. At home he is encouraged to obtain graduated exercise yet secure adequate rest. The diet may be general. There may be transitory diarrhea owing to section of the vagus nerves, but this seldom requires active treatment. The patient comes to the office on the twelfth to fifteenth postoperative day. Since these patients gain strength somewhat more slowly than others, they are advised to remain away from regular work for eight to twelve weeks.

The follow-up in these cases is extremely important. It is important not only from the standpoint of clinical evaluation of the operation but from the standpoint of the morale of the patient. Most patients experience a period of adjustment if not anxiety, which the surgeon should anticipate and endeavor to minimize by sympathetic attention and counsel. Since most of the patients have various complaints, usually minor in character, it is necessary to see them at four to six week intervals for an indefinite period.

### RESECTABILITY

In certain instances and in some types of carcinoma, extension of the tumor to lymph nodes and adjacent organs takes place early. On the other hand, many patients consult the surgeon late. These two factors more than anything else affect the rate of resectability. It appears from the literature, however, that as experience accumulates the rate of resectability is increasing. In accordance with this, my experience has been that the more operations one does, the more cases there are which prove resectable.

### MORTALITY

It appears desirable to preface remarks on results of therapy with a statement on mortality. It is important to realize in this discussion that these procedures are employed in the treatment of malignant disease, and the mortality must be considered in that light.

TABLE 1.—*Comparative Mortality in Early and Recent Experience*

| Year | Surgeon | Cases | Mortality, Per Cent |
|---|---|---|---|
| | **Early Experience** | | |
| 1871 to 1907 | All surgeons | All | 100.0 |
| 1933 | Ohsawa [13] | 43 | 53.4 |
| 1940 | Garlock [5] | 19 | 36.8 |
| 1940 | Ochsner and DeBakey [16] | 4 | 75.0 |
| 1942 | Churchill and Sweet [21] | 13 | 23.0 |
| 1943 | Phemister (Arch. Surg. **46**: 915-929 [June] 1943) | 10 | 40.0 |
| | **Recent Experience** | | |
| 1948 | DeAmesti and Otaiza (Surgery **23**: 921-934, 1948) | 14 | 57.0 |
| 1948 | Thompson (present article) | 16 | 37.5 |
| 1948 | Payne and Clagett (Surgery **23**: 912-920, 1948) | 33 | 13.0 |
| 1948 | Sweet [27] | 189 | 0.0 to 23.6 |

As recently as 1940, 40 per cent of the deaths resulting from cancer in the United States were from cancer of the esophagus and the stomach. Prior to Voelcker's report in 1908, the operative mortality was 100 per cent. It was 1913 when Torek [10] reported the first successful transthoracic resection of the esophagus. Bird [24] found reports of but 17 successful resections prior to 1938. In the early experience with these procedures, regardless of the operator, mortality ranged from 40 to 75 per cent. Likewise, in the early experience of most individual operators, the mortality is high. This was true in my experience.

The comparative mortality in the early and recent experience is given in table 1. This material is not presented as in any way critical of the individual surgeons. On the other hand, as pointed out by Garlock, it is a tribute to the perseverance, diligence and skill of the American surgeon. These figures are based on groups of cases as they are presented by the individual author. Because authors group their

cases differently, results reported are not comparable. Moreover, with the exception of Sweet,[27] the mortality presented in this table is for each author's entire group regardless of the varying mortality in his subgroups.

TABLE 2.—*Series of Cases*

| Case | Age | Diagnosis | Operation | Results |
|---|---|---|---|---|
| 1 | 43 | Adenocarcinoma of cardia with metastasis | 3/15/45; transthoracic exploration | Survived operation; died six months after operation |
| 2 | 64 | Squamous carcinoma of esophagus with metastasis | 3/30/45; transpleural exploration | Died the day after operation |
| 3 | 58 | Adenocarcinoma of cardia | 10/19/45; transthoracic cardioesophagectomy* | Died third postoperative day of massive collapse of right lung |
| 4 | 53 | Squamous cell carcinoma of mid-esophagus with extension to aorta | 1/19/46; transthoracic exploration | Survived operation; referred for roentgen therapy |
| 5 | 60 | Squamous cell carcinoma of esophagus with extension to cardia | 2/18/46; transthoracic esophagocardiectomy | Died on twenty-first postoperative day of cardiac and pulmonary complications |
| 6 | 70 | Adenocarcinoma of cardia with extension to esophagus | 3/28/46; transthoracic cardioesophagectomy | Died of shock on day of operation |
| 7 | 47 | Adenocarcinoma of cardia with extension to esophagus | 7/19/46; transthoracic cardioesophagectomy | Survived operation; died six and one-half months after operation |
| 8 | 64 | Adenocarcinoma of cardia | 7/31/46; transthoracic cardioesophagectomy | Died four days after operation of acute pulmonary edema and atelectasis |
| 9 | 54 | Adenocarcinoma of corpus with extension to cardia | 3/19/47; transthoracic cardioesophagectomy | Died eleventh postoperative day of subacute pulmonary complications |
| 10 | 42 | Adenocarcinoma of corpus with extension to cardia | 5/2/47; transthoracic cardioesophagectomy | Living eighteen months after operation with evidence of generalized carcinomatosis |
| 11 | 69 | Large benign peptic ulcer of esophagus | 7/19/47; transthoracic esophagocardiectomy | Living sixteen months postoperatively |
| 12 | 47 | Squamous carcinoma of esophagus with adenocarcinoma of cardia | 1/16/48; transthoracic esophagocardiectomy | Died eight months after operation |
| 13 | 58 | Squamous carcinoma of mid-esophagus | 2/12/48; transthoracic esophagocardiectomy | Working nine months after operation |
| 14 | 51 | Adenocarcinoma of corpus with extension to cardia | 8/13/48; subtotal gastric resection with esophagojejunostomy | Died two months after operation of carcinomatosis |
| 15 | 57 | Leiomyosarcoma of cardia | 10/20/48; transthoracic local excision of leiomyosarcoma | Survived operation; dismissed twenty-first postoperative day; returning to outpatient clinic |
| 16 | 70 | Reticulum cell lymphosarcoma of corpus with extension to cardia | 11/16/48; abdominothoracic total gastrectomy with esophagojejunostomy | In hospital fourteenth postoperative day |

* Except when specified, continuity was reestablished in all operations by esophagogastrostomy.

A series of 16 personal cases is reported in table 2. In 3 cases exploration only was possible. In 12 cases transthoracic resection was carried out, with reestablishment of continuity by esophagogastrostomy in 10 and by esophagojejunostomy in 2. In 1 case local excision of a leiomyosarcoma was performed. There were five

27. Sweet, R. H.: The Treatment of Carcinoma of the Esophagus and Cardiac End of the Stomach by Surgical Extirpation, Surgery **23**:952-975, 1948.

hospital deaths in the first series of 8 cases and 1 in the last series of 8 cases, or an over-all mortality of 37.5 per cent. One patient died two months, 1 six and one-half months and another eight months after operation. Four are living six weeks to seventeen months after operation. One patient is retired, 1 is working, 1 is ready to return to work and 1 is still in the hospital.

A review of this table indicates three things: (1) world distribution of mortality; (2) mortality according to the individual experience of the operator, and (3) the remarkable progress which has been made in the fifteen years since 1933. To sum up, the table is presented to illustrate how, beginning with a world mortality prior to 1908 of 100 per cent, by 1948 and in certain groups of cases mortality has been reduced to 0.0 per cent.

## SUMMARY AND CONCLUSIONS

During the last decade the transthoracic approach in esophageal and gastric surgery has become established. On the basis of work reported during the last few years, it appears that the abdominothoracic approach in certain cases is a valuable addition to this method of attack on lesions which are more extensive below the diaphragm. A brief discussion of the anatomy, pathology and history and of their application to the treatment of carcinoma of the esophagus, cardia, fundus and corpus of the stomach is accompanied with an outline of the clinical and diagnostic features of these conditions. Details of the preparation of the patient, anesthesiologic considerations and postoperative care have been given along with a general discussion of the transthoracic and abdominothoracic approaches and the technic of operation.

NOTE.—The patient in case 16 is living and well four months postoperatively. Since the list in table 2 was submitted, another patient has been operated on. On Dec. 8, 1949, through an abdominothoracic incision, subtotal gastrectomy with partial hepatectomy and esophagogastrostomy were performed for reticulum cell lymphosarcoma. The patient was dismissed from the hospital on the thirteenth postoperative day. Thus, in the series of 17 cases, the mortality is 35.3 per cent.

## DISCUSSION

DR. T. G. ORR, Kansas City: Some of these patients can be treated palliatively if you find the condition inoperable on exploration of the chest. I think it is well worth while. I have a patient of that type in whom an anastomosis has been made between the esophagus and the fundus of the stomach and who has been alive several months and has been able to eat rather regularly and is reasonably comfortable. In some of these cases I believe that a palliative operation may be indicated.

DR. HAROLD LINCOLN THOMPSON, Los Angeles: As Dr. Orr was speaking, it occurred to me that it should be pointed out that some of these resections obviously were palliative. One could not perform resection in the patient who lives from three to six months after operation on any other basis. It is believed, however, that the patient who is able to swallow until the last few days or weeks of life has been furnished the best palliation that any procedure yet devised has to offer.

# ANESTHESIA IN TRANSTHORACIC SURGERY OF THE ALIMENTARY TRACT

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LOS ANGELES

THE achievements of transthoracic surgery in a large measure have been made possible by the concurrent advances in anesthesiology. The major problems of anesthesia with relation to the transthoracic approach to the alimentary tract are: (1) maintenance of anesthesia throughout the period of operation; (2) provision of a quiet field for the surgeon when needed; (3) maintenance of adequate oxygenation, with aspiration of the respiratory tract when indicated; (4) maintenance of circulation with proper and adequate fluids; (5) control of vagal reflexes and treatment of cardiac arrhythmias, and (6) inflation of the lungs periodically and at the conclusion of operation. Finally, the anesthetist should work closely with the surgeon in the prevention and treatment of postoperative complications as they pertain to the respiratory tract.

The choice of anesthetic agent or agents is secondary to the ability of the anesthetist to take care of the patient during operation. While it is possible with a tight face mask to maintain anesthesia, intubation of the trachea is preferred. It not only is the best insurance against obstruction of the air passages but it provides an easy means of aspirating the lower bronchi during the operation and at its termination, and it affords greater control over the delivery of oxygen and the anesthetic gases or vapors to the alveoli. While the type of endotracheal catheter is a matter of the anesthetist's individual preference, it is of prime importance to have one large enough, usually, for adults, from 35 F to 40 F. While a tight pharyngeal pack functions well, an inflatable cuff around the endotracheal tube is preferred, to be kept inflated only when the lungs are to be expanded.

Anesthesia in transthoracic surgery demands constant vigilance and foresight, preparation for various contingencies before they arise and, above all, observation of the principle of prevention rather than treatment. Such anesthesia demands an anesthetist with an adequate knowledge of surgical physiology, especially as it pertains to the respiratory, circulatory and nervous systems and the various nervous reflexes. In

Dr. Brown has been an important member of Dr. H. L. Thompson's operating team since early 1946.

addition, he must understand the pharmacologic basis of the agents he wishes to use.

The technic of anesthesia usually is as follows: Prior to operation the surgeon discusses the general problems and the physical status of the patient with the anesthetist. The anesthetist visits the patient before the operation, appraises his physical status and questions him as to his activities with a view to determining the functional capacity of the heart or the limitations of respiratory function. The anesthetist checks the laboratory data, especially the red blood cell and the hemoglobin counts. The anesthetist writes the hospital orders for premedication. In the average case pentobarbital sodium, 3 grains (0.19 Gm.), is given by mouth at bedtime. Phenobarbital sodium, 2 grains (0.13 Gm.), is given intramuscularly two hours preoperatively after the Levin tube is in place. Morphine sulfate, 1/6 grain (0.01 Gm.), and scopolamine, 1/150 grain (0.0004 Gm.), are administered one hour preoperatively.

When the patient arrives at the operating room, an intravenous drip of one-sixth molar sodium *r*-lactate is started and anesthesia is induced with pentothal sodium® and *d*-tubocurarine chloride solution.® During the induction period and while the effect of previously administered medicaments is being observed, the patient is given a mixture of 75 per cent nitrous oxide with 25 per cent oxygen. As the curare takes its effect and the respiratory exchange is decreased, the lungs are inflated by manual compression of the breathing bag. This constitutes compensated or assisted respiration given to avoid hypoxia. Thus, after a period of three to five minutes, the patient may be intubated under direct vision laryngoscopy. After this anesthesia is maintained with nitrous oxide and oxygen in a semiclosed system, with frequent manual assistance to respiration after the pleura is opened. Small amounts of pentothal sodium® may be given as indicated. The concentration of oxygen is increased as indicated. Demerol hydrochloride® may be used intravenously if it is apparent that excessive amounts of pentothal sodium® are required.

Every twenty to thirty minutes the progress of the operation is interrupted for one to two minutes while the collapsed lung is expanded by the anesthetist. Prolonged collapse of the lung is undesirable because it produces changes in the pulmonary capillaries as a result of altered blood flow as well as changes in the alveoli themselves. Whenever mucus or other secretion accumulates, it is desirable to aspirate the trachea before inflating the lung.

In all cases a vein is cannulated with an 18 gage needle for administration of blood, crystalloid, pentothal sodium,® procaine hydrochloride or curare. Infusion of compatible blood is started when the cutaneous

incision is made and blood is replaced as rapidly as it is lost. In cases of impaired physical status or low hemoglobin content it is essential to have two veins cannulated so that blood may be pumped in rapidly if needed. To one of the needles I always have a three way stopcock attached. Through one of these, intravenous drip is maintained for administration of the various solutions mentioned. In addition, 5 per cent dextrose in water or sodium chloride solution is used to keep the intravenous needle patent. Each patient receives 500 cc. of sixth molar sodium *r*-lactate preliminary to blood transfusion in order to alkalinize the blood and as prophylaxis against transfusion reaction. Since as a rule shock is synonymous with loss of blood or rough handling of tissues, it is most important to replace blood with blood as it is lost rather than with plasma or with crystalloid solutions. Moreover, it should be replaced at the time it is lost and not afterward. Only by scrupulous attention to many such details can the surgical team expect to maintain the same blood pressure, pulse rate and pulse volume throughout the course of operation.

Apace with the increase in intrathoracic surgery are the number of reports of cardiac arrhythmia and of cardiac standstill. Much remains to be learned about these phenomena. The danger of arrhythmias lies in the progress of certain types to a downward displacement of the pacemaker amounting to ventricular tachycardia followed by ventricular fibrillation, which usually is fatal. The surgeon and the anesthetist must be on a constant watch for cardiac arrhythmia.

In some reported cases it has seemed desirable as prophylaxis to inject into the autonomic nerves at the hilus of the lung 1 per cent procaine hydrochloride either before section of the vagi or immediately arrhythmia is noted. In some instances I have given as prophylaxis an intravenous drip of procaine hydrochloride, 0.1 to 0.5 per cent in dextrose, water or sodium chloride solution. According to reports, the unconscious patient tolerates large doses of intravenously administered procaine hydrochloride without untoward effect and without the appearance of the characteristic symptoms expected on administration of the same dose to the conscious patient. Amounts up to 10 Gm. during an operation of several hours' duration have been reported in the literature to have produced no untoward effect.

If arrhythmia occurs suddenly, one may give procaine hydrochloride, 50 to 100 mg. intravenously, as the most rapid means of administration.

While it is not always necessary for the surgeon to discontinue operation, he should do so if it is indicated. As an alternative he may transfer his activities temporarily to another area of the operative field. If cardiac stoppage occurs, the surgeon should immediately institute manual compression of the heart until spontaneous heart beat is resumed within thirty to sixty seconds. If heart beat is not resumed within this

time, he should inject 1.0 cc. of epinephrine hydrochloride directly into the heart. While the use of epinephrine is viewed as debatable by some, reports indicate that it has promptly restored cardiac activity in patients who otherwise probably would have died. During this trying period, the anesthetist keeps the patient well oxygenated by manual compression of the breathing bag, maintains the blood volume and administers procaine hydrochloride intravenously. A plan of action should be agreed on by the anesthetist and the surgeon before this serious complication is encountered.

It hardly seems necessary to state that it is a part of the anesthetist's duty to keep constantly aware of what the surgeon is endeavoring to do. When the lung projects into the field of operation, the anesthetist can deflate it temporarily and thus greatly facilitate the working conditions of the surgeon. This control of pulmonary activity can be achieved either by controlled respiration technic or by the use of curare to paralyze the respiratory muscles. Properly spaced doses of curare relax the abdominal muscles and prevent the upward pressure of the abdominal contents against the diaphragm, which interferes with the surgeon's work. Judicious use of curare will also weaken diaphragmatic activity without paralyzing it. Efforts directed toward improving the working conditions of the surgeon expedite operation and thus shorten the duration of anesthesia and the surgical procedure.

When the pleura is being closed, the anesthetist inflates the lung. He should do so just short of bringing the lung to the periphery of the thorax so as not to interfere with the use of the suture needle. This precaution prevents injury to the visceral pleura.

At the conclusion of operation, the lung is expanded under direct vision. When the wound in the chest is closed and is air tight, the drainage tube from the pleural space is placed under water at a level lower than the patient. During this time the lungs are further inflated while the anesthetist alters the position of the patient by elevating or lowering the head of the operating table and by turning the patient from side to side so as to force all the air out of the pleural space. Finally, the trachea then is aspirated before the endotracheal tube is removed. By careful manipulation of the anesthetic agent, one can have the patient awake or at least in a state wherein the swallowing reflex is active before he is removed from the operation table. This decreases the amount of immediate nursing care required and lessens the likelihood of pulmonary atelectasis.

The anesthetist's participation in the early postoperative care of the patient is as important as it is during operation. During this period the anesthetist is concerned chiefly with the prevention and treatment of atelectasis. To this end the patient's chest must be examined every six to eight hours for the first two or three days. While an unexplained

rise in pulse, respiratory rate and temperature and the advent of cyanosis may be significant, these indicators follow rather than antedate the onset of atelectasis. Thus at the time the patient is seen, these indicators may be normal. We place great dependence on suppressed breath sounds over the involved area. The important thing to remember is that the physical signs are not striking in early or minimal atelectasis.

A pulse rate over 100 per minute with slight alteration of breath sounds is sufficient for a presumptive diagnosis of atelectasis in the absence of other causes. As treatment tracheobronchial aspiration is employed under topical anesthesia with pontocaine hydrochloride® in 2 per cent solution. A urethral catheter, size 18 F or 20 F, is passed through the nose into the trachea either blindly or under direct vision. Contact of the catheter with the carina produces violent coughing. Then, by connection of the catheter with the aspirating machine for fifteen to thirty seconds, mucus may be brought up. After the coughing has ceased, oxygen is administered for sixty to ninety seconds, after which aspiration is repeated until all mucus is removed.

In no other type of surgery is teamwork and cooperation between the anesthetist and the surgeon so important. Moreover, the importance of careful observation and prophylactic treatment cannot be overemphasized.

# TREATMENT OF JEJUNAL ULCER

## A Comparative Follow-Up Study

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RIENHOFF,[1] in discussing the surgical treatment of gastroduodenal ulceration, stated that, regardless of the surgeon or the type of operation performed, when gastric and jejunal mucosa are joined in the presence of acid chyme a certain number of patients will suffer from postoperative complications similar to the original complications of the primary ulcer for which the operation was performed. The truth of this statement has been convincingly demonstrated in appraisals of the remote results of gastroenterostomy, and in fact the frequency of recurrent jejunal ulcer following this operation has been the chief reason for the almost general abandonment of it. Gastric resection, moreover, while yielding results superior to those of gastroenterostomy, is attended by a definite although small percentage of failures due to the development of a secondary ulcer at or near the gastroenteric stoma. The duodenal exclusion operations, in the opinion of most surgeons, are the least satisfactory of all procedures for this same reason.

The incidence of jejunal ulcer following gastroenterostomy has been variously estimated by different authors. In the earlier years, when this procedure was at the height of its popularity, this incidence was estimated by Judd and Hoerner[2] as 2.8 per cent, by Benedict[3] as 2.9 per cent, by Balfour[4] as 3.26 per cent and by Wilkie[5] as 3.5 per cent. When the end results are evaluated many years later, it becomes apparent that the actual incidence is considerably higher than these earlier figures would indicate. Thus, in recent years, Ogilvie[6] esti-

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1. Rienhoff, W. F.: An Analysis of the Results of Surgical Treatment of Two Hundred and Sixty-Six Cases of Chronic Peptic Ulcer of the Duodenum, Ann. Surg. **121**:583, 1945.

2. Judd, E. S., and Hoerner, M. T.: Jejunal Ulcer, Ann. Surg. **102**:1003, 1935.

3. Benedict, E. B.: Jejunal Ulcer: An Analysis of Thirty-Six Cases and Study of the Literature, Surg., Gynec. & Obst. **56**:807, 1933.

4. Balfour, D. C.: Results of Gastroenterostomy, Ann. Surg. **92**:558, 1930.

5. Wilkie, D. P. D.: Jejunal Ulcer: Some Observations on Its Complications and Their Treatment, Ann. Surg. **99**:401, 1934.

6. Ogilvie, W. H.: The Place of Surgery in the Treatment of Peptic Ulcer, Lancet **1**:419, 1935.

mated the incidence to be about 20 per cent, Lahey [7] 15 per cent or higher and Wangensteen [8] 15 per cent. Cooper,[9] in a critical review of 279 gastroenterostomies performed at the New York Hospital over a thirteen year period, reported an operative mortality of 3.2 per cent and a recurrence rate of 25.2 per cent among the survivors during the first five years. However, he pointed out that reevaluation at the end of the five year period revealed that one quarter of these patients had become relieved by further medical treatment, and the percentage of failures is therefore reduced to 16.3 per cent. He concluded that a patient subjected to gastroenterostomy has a 96.7 per cent chance of surviving the operation and, if he does survive, he has a 78 per cent chance of remaining well thereafter. Even though he should have further trouble, he has an 88 per cent chance of becoming well without further surgical treatment and a 92 per cent chance of being relieved should further surgical intervention be required. In other words, his chances of survival and ultimate cure are about 89 per cent. Gardner and Hart [10] have recently reported on a group of 68 patients carefully selected and treated with gastroenterostomy. There were 5 deaths, or a mortality of 7.3 per cent, and an incidence of recurrence of 5.6 per cent. They stated the belief that gastroenterostomy is a satisfactory operation provided it is restricted to a certain group of patients, i. e., those in the older age groups with relatively inactive ulcers and a high degree of pyloric obstruction.

With regard to gastric resection, Allen and Welch [11] reported three jejunal ulcers in 129 primary operations for duodenal ulcers (Finsterer operations excluded), or an incidence of 2.3 per cent. St. John, Harvey, Ferrer and Sengstaken [12] reported 2 cases of verified jejunal ulcer among 373 patients who survived operation and 3 additional cases in which jejunal ulcer may have occurred. The incidence was, therefore, somewhere between 0.5 and 1.4 per cent. Wangensteen reported only 2 instances of soma ulcer in over 500 consecutive resections for ulcer

7. Lahey, F. H.: Inflammatory Lesions of the Stomach and Duodenum, J. A. M. A. **127**:1030 (April 21) 1945.

8. Wangensteen, O. H.: The Role of the Surgeon in the Management of Peptic Ulcer, New England J. Med. **236**:191, 1947.

9. Cooper, W. A.: End Results in the Treatment of Peptic Ulcer by Posterior Gastroenterostomy, Surgery **23**:425, 1948.

10. Gardner, C. E., and Hart, D.: The Surgical Treatment of Peptic Ulcer: A Comparison of the Results of Gastroenterostomy, Gastric Resection and Vagotomy at the Duke Hospital, Ann. Surg. **127**:1056, 1948.

11. Allen, A. W., and Welch, C. E.: Subtotal Gastrectomy for Duodenal Ulcer, Ann. Surg. **124**:688, 1946.

12. St. John, F. B.: Harvey, H. D.; Ferrer, J. M., and Sengstaken, R. W.: Results Following Subtotal Gastrectomy for Duodenal and Gastric Ulcer, Ann. Surg. **128**:3, 1948.

(0.4 per cent). Lahey estimated the incidence of jejunal ulcer to be from 2 to 3 per cent after subtotal gastrectomy. Gardner and Hart reported 123 gastric resections (including Finsterer operations) with an operative mortality of 8.9 per cent. Among the 100 cases which were followed there were 5 recurrences.

In most clinics the results achieved by the duodenal or antral exclusion operations advocated by Devine [13] and Finsterer [14] have been disappointing. Ogilvie [15] estimated that with exclusion gastrectomy jejunal ulcer may be anticipated in 30 to 40 per cent of cases. In his series of Finsterer resections for exclusion performed in 1937, a jejunal ulcer developed within two years in 9 of 22 patients and in 1 instance a jejunal ulcer perforated in less than three weeks after the patient left the hospital. Lahey stated that with 20 Finsterer operations performed in his clinic a higher incidence of anastomotic ulcer in the stump of the resected stomach was observed than with any other procedure. Allen [16] reported that jejunal ulcer developed in 5 of his 9 cases in which Finsterer resection was done. Three of the patients were reoperated on and cured by antrumectomy and resection of the ulcer area. In contrast with this, he had performed exclusion resections in 20 cases in which the antral mucosa was removed as recommended by Bancroft,[17] with no jejunal ulcers. He further observed 2 patients with jejunal ulcer, originally operated on elsewhere by the Finsterer technic, in whom there was prompt recurrence. Both of these patients were relieved by antral excision only. In an experience with nearly 100 cases in which the antrum was left but the mucosa removed, Wangensteen found no jejunal ulcers. Gardner and Hart stated that in 23 of their cases of resection a duodenal ulcer was excluded by dividing the pyloric antrum 6 to 8 cm. above the pylorus. In 12 cases the antral mucosa was removed, and in 11 it was left intact. There was only one recurrent ulcer in this group of 23 cases, and in this case the pyloric mucosa had been left. They stated the belief that antral exclusion is a useful procedure but now recommend exenteration of the antral mucosa.

As yet it is too early to determine the position that vagotomy will eventually come to occupy in the management of duodenal ulcer. Being

13. Devine, H. B.: Basic Principles and Supreme Difficulties in Gastric Surgery, Surg., Gynec. & Obst. **40**:1, 1925.

14. Finsterer, H.: Ausgedehnte Magenresektion bei Ulcus duodeni statt der einfachen Duodenal-resektion bzw., Pylorusausschaltung, Zentralbl. f. Chir. **45**: 434, 1918.

15. Ogilvie, H.: Gastrectomy: A Human Experiment, Lancet **2**:377, 1947.

16. Allen, A. W.: The Importance of the Antral Mucosa in the Surgical Treatment of Peptic Ulcer, South. M. J. **36**:368, 1943.

17. Bancroft, F. W.: A Modification of the Devine Operation of Pyloric Exclusion for Duodenal Ulcer, Am. J. Surg. **16**:223, 1932.

frequently combined with gastroenterostomy as it is today, it will, of necessity, require many years before a final evaluation can be made. Certain it is that jejunal ulcer occasionally develops after this dual procedure (Wilkinson [18]). A sufficient number of jejunal ulcers have been recorded to indicate that recurrence of this type is a real threat when gastroenterostomy is used in combination with vagotomy.

The present study is concerned with a group of 81 patients treated surgically for postoperative jejunal ulcer at the University Hospital during the past twenty years. Operations performed for gastrojejunocolic fistula or for a malfunctioning or unnecessary gastroenterostomy opening as well as those for recurrent duodenal ulcer following plastic procedures on the pylorus have been excluded intentionally.

It was hoped to determine, if possible, the factors responsible for the failures of surgical treatment, the time of recurrence in relation to the primary operation and whether such patients might ultimately be made well by further surgical treatment and, if so, which of the available procedures were most likely to succeed. Of the 81 patients, 69, or 85.2 per cent, were men and 12, or 14.8 per cent, were women. The age range was 25 (3 patients) to 75 years, with an average of 48.4 years. The age range for the female patients was 39 to 70 years, with an average of 51.4 years. The average for the male patients was 47.9 years. This group of patients had proved to be unusually refractory to the usual forms of treatment, and already they had undergone a total of 106 previous operations for some type of peptic ulceration. One patient had been subjected to four previous operations, 3 patients to three operations, 16 patients to two operations and the remaining 61 to one operation. A list of these previous operations is given in table 1. It will be noted that gastroenterostomy was the procedure which most often had failed, and this was followed in frequency by the duodenal exclusion operations, while gastric resection, including three pylorectomies, was third in importance. The list is largely self explanatory. In 2 cases a gastric ulcer was excised locally. Subsequently, gastric retention of a degree sufficient to require gastroenterostomy developed in both patients. In 2 patients an initial gastroenterostomy mechanism had already been dismantled and later reestablished. A jejunojejunostomy had been performed without success in 1 case for a jejunal ulcer which had followed gastroenterostomy. The gastrostomy and the subsequent closure were employed in the treatment of an esophageal ulcer producing severe dysphagia in a patient who previously had undergone a gastroenterostomy for a duodenal ulcer. Thirty-two of the 106 original operations were performed

18. Wilkinson, S. A.: The Peptic Ulcer Problem, J. A. M. A. **138**:805 (Nov. 13) 1948.

in University Hospital, and the operation directly responsible for the jejunal ulcer was performed elsewhere in 53 instances and at this hospital in 28. These operations are divided into the three aforementioned groups: (1) gastroenterostomy in 57 instances; (2) gastric resection in 9, and (3) antral exclusion operations in 15.

It is commonly believed that antecolic anastomoses, with a long proximal jejunal loop, are especially apt to be followed by recurrence (Lannin [19]) and that this is particularly true if an enteroenterostomy is added. In this series only 2 of the gastroenterostomies were of the anterior type, while 55 were of the retrocolic variety, and in no instance

TABLE 1.—*Previous Operations—81 Patients*

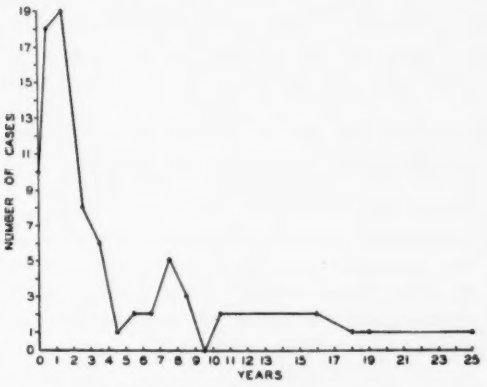
| Surgical Procedure | No. of Operations |
|---|---|
| Gastroenterostomy | 60 |
| Devine exclusion | 8 |
| Gastric resection | 7 |
| Closure of acute perforation | 7 |
| Finsterer resection for exclusion | 6 |
| Pylorectomy | 3 |
| Excision of gastric ulcer | 2 |
| Closure of acute perforation and gastroenterostomy | 2 |
| Dismantling of gastroenterostomy mechanism | 2 |
| Gastroenterostomy and excision of duodenal ulcer | 1 |
| Vagotomy (abdominal) | 1 |
| Plastic to jejunal ulcer | 1 |
| Jejunojejunostomy | 1 |
| Gastrostomy | 1 |
| Closure of gastrostomy | 1 |
| Unknown | 3 |
| Total | 106 |

had complementary enteroenterostomy been performed. In the group of 9 cases in which gastric resection had failed, small resections which were scarcely more than pylorectomies had been performed in 2, while in the remaining 7 one half or more of the stomach had been removed. In these 7 cases the reconstruction had been made by the posterior Polya method in 3, the anterior Polya method in 1 and the posterior Hofmeister method in 2, all without enteroenterostomy. In 1 case the technic employed originally was not ascertained, as the patient was treated by means of transthoracic vagotomy. In the case of the two pylorectomies, 1 patient had undergone antecolic anastomosis and 1 a posterior Roux Y type of anastomosis, a procedure generally conceded to be conducive to a poor result.

19. Lannin, B. G.: Evaluation of a Satisfactory Operation for Ulcer, Surgery **17**:712, 1945.

The exclusion operations consisted of the Devine procedure (simple transection through or above the antrum and reconstruction without the resection of any gastric tissue) in 8 instances and Finsterer's resection for exclusion in 7. In all cases the Devine procedure that had been carried out was posterior anastomosis without enteroenterostomy. Of the Finsterer operations, five had consisted in posterior anastomosis and two in antecolic connection. In 1 case in which the latter was performed a complementary enteroenterostomy had been added. All these exclusion operations, save one Devine procedure, had been performed in this hospital.

The total experience with the exclusion operations may be summarized briefly in the following statements. Follow-up data are available




Interval between responsible operation and recurrence of symptoms.

in 22 cases of primary duodenal ulcer treated by Finsterer's resection for exclusion, 14 similar cases in which the Devine exclusion operation was performed and 29 cases in which the Finsterer procedure modified by exenteration of the antral mucosa was employed. The results in these three groups are: (*a*) 7 patients are well, 5 are improved and 10 are in poor condition; (*b*) 2 patients are well, 3 are improved and 9 are in poor condition; (*c*) 25 patients are well, 1 is improved and 3 are in poor condition.

With regard to the lesion for which the original operations were performed, it is worthy of mention that none of these procedures was for gastric neoplasms, e. g., carcinoma, leiomyoma, neurofibroma, lymphosarcoma or polyposis. In 7 cases the lesion was a gastric ulcer, and in the remainder the evidence is either positive or presumptive that the ulcer was distal to the pylorus.

The accompanying graph presents data concerning the interval between the responsible operation and the onset of symptoms of recurrence. It is somewhat surprising to note that in 10 cases recurrent symptoms appeared almost immediately, i. e., before or shortly after the patient left the hospital. In 18 additional cases recurrence took place in less than one year, and in 19 others it had manifested itself in less than two years. Thus, in 34.6 per cent of the patients recurrence had taken place in less than one year, in 58.6 per cent in less than two years and in 76.5 per cent within five years. While in most cases the recurrence was observed by the end of five years, it will be observed that in 2 it was observed at sixteen years, in 1 at eighteen, in 1 at nineteen years and in 1 at twenty-five years.

The indications for further surgical intervention in these cases of jejunal ulcer were chiefly pain and hemorrhage, either singly or in combination. Forty-three patients had predominantly symptoms of ulcer pain, 25 pain and hemorrhage and 7 bleeding without other significant symptoms. Two patients in this last group exhibited severe anemia due to loss of blood. Five patients were found to have considerable gastric retention due to duodenal stenosis consequent on the original ulcer and subsequent closure of the gastroenteric stoma by the jejunal ulcer. In 1 case a jejunal ulcer which had followed a posterior gastroenterostomy underwent acute perforation. All patients for whom a diagnosis of jejunal ulcer was made have not been treated surgically, and some have remained reasonably well on a strict medical regimen. However, the ever present danger of the development of a gastrojejunocolic fistula makes one apprehensive of long delay if evidence of improvement is not soon demonstrable.

## TREATMENT

When dealing with jejunal ulcer, the operation chosen is governed considerably by the previous operative procedure, the condition of the patient, the technical difficulties presented and, in part, the preference of the surgeon. In the discussion of this phase of the problem the grouping used previously has been followed.

### JEJUNAL ULCER FOLLOWING GASTROENTEROSTOMY

Cases of jejunal ulcer following gastroenterostomy constituted the largest group, numbering 57 (table 2). Considered from a historical viewpoint, the favored procedures have been: (1) dismantling of the gastroenterostomy mechanism, with or without a plastic procedure on the pylorus; (2) some form of gastric resection, and (3) vagotomy.

*Dismantling of Gastroenterostomy Mechanism.*—When gastroenterostomy has failed it was believed formerly that restoration of the

gastrointestinal tract to normal afforded the safest and best solution. Inasmuch as the original ulcer had usually healed at this time, symptoms often could be controlled reasonably well by restoration of the tract to normal and elimination of the anastomotic ulcer, to be followed by a careful medical regimen. In certain cases in which an original gastroenterostomy had been performed without adequate indication this was especially true. If narrowing of the pyloric orifice due to scar was found to persist, enlargement could be readily effected by an associated pyloroplasty. Simple dismantling of the gastroenterostomy mechanism was performed in 10 cases in this series, with 2 operative deaths, 1 due to peritonitis (autopsy) and 1 due to intestinal obstruction (no autopsy). Disconnection of the anastomosis, with a complementary pyloroplasty, was carried out in 8 cases, with no deaths. These procedures are simpler and safer than those involving some form of gastric resection

TABLE 2.—*Treatment of Jejunal Ulcer Following Gastroenterostomy—57 Cases*

| Operation | No. of Cases |
|---|---|
| Gastric resection | 32 |
| Dismantling of gastroenterostomy mechanism (2 deaths) | 10 |
| Dismantling of gastroenterostomy mechanism and pyloroplasty | 8 |
| Finsterer exclusion | 3 |
| Finsterer-Bancroft exclusion | 1 |
| Excision of ulcer with Reanastomosis | 1 |
| Gastric resection and abdominal vagotomy | 1 |
| Jejunostomy | 1 |

and avoid the danger of the formation of a new stomal ulcer, although the possibility of recrudescence of the original ulcer or the formation of a new one in the stomach or duodenum remains.

*Gastric Resection.*—With the available evidence indicating the effectiveness of partial or subtotal gastrectomy in the treatment of primary gastric or duodenal ulcer, logically it should promise superior results in the treatment of jejunal ulcer. Subtotal resection as a secondary operation at times entails considerable risk, and the technical difficulties associated with the removal of a large inflammatory mass at the stoma may necessitate the selection of some other procedure. Standard resection with appropriate management of the involved jejunal segment was employed in 32 cases, and Finsterer's resection for exclusion was used in 3. In these 3 cases the pyloric antrum was left intact, whereas in an additional case a Finsterer operation was performed and the antral mucosa removed.

*Local Excision.*—In 1 case the jejunal ulcer was excised locally and the original anastomosis reconstructed.

*Jejunostomy.*—In 1 debilitated patient with severe bleeding from a jejunal ulcer and a large inflammatory mass at the stoma a jejunostomy for feeding was employed, with the hope that as a result of physiologic rest subsidence would take place.

*Vagotomy.*—In this group solitary vagotomy as the first operative procedure for jejunal ulcer was not performed. In 1 case subdiaphragmatic section of the vagus nerve was employed in addition to gastric resection.

### JEJUNAL ULCER FOLLOWING GASTRIC RESECTION

When a classic type of resection, including removal of the antrum, had already been performed, the several procedures consisted of reresection of the stomach, local excision of the ulcer and vagotomy (table 3).

*Reresection.*—When an original resection has failed, resection higher up would seem likely to succeed in view of the clinical and experimental evidence presented by Wangensteen, who stated the belief that a three-quarter resection, with a short afferent loop, is essential for a

TABLE 3.—*Treatment of Jejunal Ulcer Following Gastric Resection—9 Cases*

| Operation | No. of Cases |
|---|---|
| Reresection (1 death) | 7 |
| Excision of ulcer with reconstruction of anastomosis | 1 |
| Transthoracic vagotomy | 1 |

satisfactory result. Since many of the original resections had been of less magnitude than this, the performance of a large or adequate resection was undertaken in 7 cases. There was 1 death in this group due to postoperative hemorrhage (no autopsy).

*Local Excision.*—In 1 case a simple excision of the anastomotic ulcer, with repair of the old anastomosis, was performed.

*Vagotomy.*—In 1 case a transthoracic vagotomy was elected.

### JEJUNAL ULCER FOLLOWING EXCLUSION OPERATIONS

In the group of cases of jejunal ulcer following exclusion operations, because of the anatomic features and the physiologic principles involved, several procedures may be considered. These are (1) reresection with excision of the antrum; (2) reresection without antral excision; (3) antral excision only, and (4) vagotomy (table 4).

*Reresection with Antral Excision.*—Although the most comprehensive and not always technically feasible, this would seem to be the preferable procedure. It was employed in 6 cases, with no deaths.

*Reresection Without Antral Excision.*—In view of the accumulated evidence regarding the danger attendant on a retained pyloric antrum,

this procedure might be expected to invite recurrence. It was utilized in 4 cases, with 1 death, probably due to peritonitis (no autopsy).

*Antral Excision Only.*—The interesting observation has been made by McKittrick,[20] Marshall,[21] Allen, Lahey and others that jejunal ulcers may undergo prompt healing after the removal of a retained antrum. In this series antral excision only was employed in 3 cases, with 1 death, which was the result of pancreatitis and peritonitis (autopsy) presumably due to injury to the pancreas during the dissection in a field of dense adhesions. The closed duodenal stump was found to be intact when examined at autopsy.

*Vagotomy.*—In the extensive literature now available concerning vagotomy, the results reported from its use in the treatment of recurrent ulcer following resection are remarkable. Dragstedt [22] has reported cure by vagotomy in a case of recurrence following a Finsterer exclusion procedure even though the antrum remained. Moore,[23] however, observed that transthoracic vagotomy alone failed to cure but that when

TABLE 4.—*Treatment of Jejunal Ulcer Following Exclusion Operations—15 Cases*

| Operation | No. of Cases |
|---|---|
| Resection with antral excision | 6 |
| Resection without antral excision (1 death) | 4 |
| Antral excision only (1 death) | 3 |
| Antral excision and transthoracic vagotomy (two stage) | 1 |
| Jejunostomy (1 death) | 1 |

the antrum was eliminated recovery promptly took place. The experience of my colleagues and me in this field is limited to 1 case in which a combined procedure was used. Antral excision was performed as a first stage and was followed three weeks later by a transthoracic vagotomy.

*Jejunostomy.*—In 1 case, because of almost complete obstruction at the stoma due to a recurrent ulcer in a patient who originally had undergone a Finsterer operation, jejunostomy only was performed.

In table 5 the operative deaths are summarized. There were 6 deaths among the 81 operations, or an over-all mortality of 7.4 per cent.

20. McKittrick, L. S.; Moore, F. D., and Warren, R.: Complications and Mortality in Subtotal Gastrectomy for Duodenal Ulcer: Report on Two Stage Procedure, Ann. Surg. **120**:531, 1944.

21. Marshall, S. F., cited by McKittrick, Moore and Warren.[20]

22. Dragstedt, L. R.; Harper, P. V.; Tovee, E. B., and Woodward, E. R.: Section of the Vagus Nerves to the Stomach in the Treatment of Peptic Ulcer: Complications and End Results After Four Years, Ann. Surg. **126**:687, 1947.

23. Moore, F. D.: Vagus Resection for Ulcer: An Interim Evaluation; II. Clinical Results, Ann. Surg. **126**:664, 1947.

It is noteworthy that a number of the deaths occurred after the simpler procedures. This probably is to be explained by the fact that such procedures were usually chosen for the poor risk patients and that many of these operations were performed during the earlier years when the preoperative and postoperative management of fluid and electrolyte balance was poorly understood and before the advent of chemotherapeutic and antibiotic agents and the institution of blood banks.

### FOLLOW-UP STUDIES

Seventy-five patients survived operation and have been followed to the time of their death, from whatever cause, or until the completion of this report. Table 6 gives the duration of the follow-up study in five

TABLE 5.—*Operative Deaths (6; Mortality, 7.4 Per Cent)*

| Operation | Cause of Death | Autopsy |
|---|---|---|
| Dismantling of gastroenterostomy mechanism..... | Peritonitis (leak at closure of stomach) | + |
| Dismantling of gastroenterostomy mechanism..... | Intestinal obstruction | — |
| Resection ..... | Hemorrhage | — |
| Resection ..... | Peritonitis (?) | — |
| Antral excision..... | Pancreatitis and peritonitis | + |
| Jejunostomy ..... | Inanition | — |

TABLE 6.—*Duration of Follow-Up Study—75 Cases*

| Number of Years After Operation | Living | Dead |
|---|---|---|
| 5 or less..... | 25 | 15 |
| 5 to 10..... | 19 | 5 |
| 10 to 15..... | 4 | 2 |
| 15 to 20..... | 1 | 1 |
| Over 20..... | 3 | .. |
| Total..... | 52 | 23 |

year periods. The 52 patients still living have been followed for from one to twenty-three years. In the case of the 23 patients who have died since their discharge from the hospital, data regarding the cause of death are found in table 7. While the usual causes of death in persons of this age, e. g., coronary thrombosis, pneumonia and hypertension, are in evidence, it is disturbing to find that 14 patients, or 61 per cent, died of further recurrence. Nine of these deaths were due to a new recurrent ulcer and for the most part were accounted for by the complications of hemorrhage and perforation. The remaining 5 patients died after secondary operations for recurrent or jejunal ulcer. Further details of this group are given later.

In the final evaluation of results, each patient was classified on the basis of all the data collected over the years. So far as possible the results listed are in terms of symptoms of ulcer, complications and related disability. Other factors, such as associated diseases or senility, are excluded. The term "well" indicates that the patient is free from symptoms of ulcer, e. g., pain, nausea and vomiting or a history of bleeding, and that he is on an unrestricted or a slightly restricted diet. "Improved" indicates that there are some minor residual symptoms, e. g., pain or discomfort at times, especially after dietary indiscretions. Such symptoms are not incapacitating and are controlled with a reasonable amount of care. In this group there is no roentgenologic or gastroscopic evidence of recurrence. A "poor" result indicates that the patient remains unimproved or has become worse. Patients with poor results have uniformly shown evidence of recurrence, either in the form

TABLE 7.—*Cause of Death in 23 Patients Who Have Died Since Discharge*

| Cause of Death | No. of Cases |
|---|---|
| Recurrent ulcer | 9 |
| Reoperation for recurrence | 5 |
| Coronary thrombosis | 3 |
| Pneumonia | 2 |
| Hypertension and myocarditis | 2 |
| Cerebral hemorrhage | 1 |
| Accident | 1 |

of a new recurrent jejunal ulcer after resection or exclusion operations or as a new gastric or duodenal ulcer or the reactivation of an old one when the gastrointestinal tract was restored to normal. None of the results listed as poor has been due to complications of the operation, such as injury to the bile ducts, intestinal obstruction, fistula or ventral hernia, or to side effects such as the dumping stomach or serious postresection symptoms.

### JEJUNAL ULCER FOLLOWING GASTROENTEROSTOMY

In table 8 data are presented concerning the results of the several operations for ulcers following gastroenterostomy (55 cases). As might be anticipated, gastric resection gave the best results, since 24 of the 32 patients treated by this method were entirely well and symptom free. One patient was improved, whereas in 7 the results were poor, in 3 instances sufficiently to require further surgical treatment. All patients who required additional surgical treatment are considered in detail in following paragraphs. In 8 cases in which simple closure of a gastroenterostomy opening was performed, 3 patients became well and 1 was improved, while in 4 the results were poor. Two of these

4 patients required further surgical treatment. Closure of the gastroenterostomy opening combined with pyloroplasty resulted in a smaller number of patients who became well (only 1), a higher incidence of those who were improved (6) and only 1 poor result. The patient who had this result required further surgical therapy.

Contrary to our expectations, the Finsterer exclusion operations resulted in failure in only 1 instance, the patient requiring further surgical treatment, while the 2 remaining patients became symptom free. The results in the case in which Bancroft's modification of the Finsterer procedure was employed was entirely satisfactory. Local excision of the ulcer with reestablishment of the original gastroenterostomy opening

TABLE 8.—*Results of Treatment of Ulcers Following Gastroenterostomy—55 Cases*

| Type of Operation | Result | No. of Cases |
|---|---|---|
| Gastric resection | Well | 24 |
| | Improved | 1 |
| | Poor | 7 (additional surgical treatment — 3) |
| Dismantling of gastroenterostomy mechanism | Well | 3 |
| | Improved | 1 |
| | Poor | 4 (additional surgical treatment — 2) |
| Dismantling of gastroenterostomy mechanism and pyloroplasty | Well | 1 |
| | Improved | 6 |
| | Poor | 1 (additional surgical treatment — 1) |
| Finsterer exclusion operation | Well | 2 |
| | Poor | 1 (additional surgical treatment — 1) |
| Finsterer-Bancroft exclusion operation | Well | 1 |
| Excision of ulcer with reanastomosis | Poor | 1 |
| Gastric resection and abdominal vagotomy | Well | 1 |
| Jejunostomy | Poor | 1 |

gave a poor result since symptoms promptly returned. The patient treated with a combined classic resection and abdominal vagotomy obtained an excellent result. The palliative jejunostomy is recorded as a failure, as the patient died four months after operation of massive hemorrhage from the unhealed ulcer.

### JEJUNAL ULCER FOLLOWING RESECTION

In the small group of 8 cases in which a primary gastric resection had failed, further resection of the stomach afforded an excellent result in 4 cases and a poor result in 1, while 1 patient was improved. As in the gastroenterostomy group, the local excision of a stomal ulcer with reestablishment of the same anastomosis resulted in failure. The one example of a solitary transthoracic vagotomy was followed by an excellent result (table 9).

JEJUNAL ULCER FOLLOWING EXCLUSION OPERATIONS

In the group of 12 cases of ulcer following exclusion operations, the procedure of greatest magnitude, i. e., reresection and excision of the antral segment, was employed in 6 cases, and all the patients were restored to excellent health. Further resection of the stomach not accompanied with removal of the antrum was, on the whole, unsuccessful, since only 1 patient among 3 became well, 1 was improved and in 1 the result was so poor that further surgical treatment was necessary. In our experience treatment of anastomotic ulcers following exclusion

TABLE 9.—*Results of Treatment of Ulcers Following Gastric Resection—8 Cases*

| Type of Operation | Result | No. of Cases |
|---|---|---|
| Reresection | Well | 4 |
| | Improved | 1 |
| | Poor | 1 |
| Excision of ulcer with reanastomosis | Poor | 1 |
| Transthoracic vagotomy | Well | 1 |

TABLE 10.—*Results of Treatment of Ulcers Following Exclusion Operations —12 Cases*

| Type of Operation | Result | No. of Cases |
|---|---|---|
| Reresection with antral excision | Well | 6 |
| Reresection without antral excision | Well | 1 |
| | Improved | 1 |
| | Poor | 1 (additional surgical treatment — 1) |
| Antral excision and transthoracic vagotomy | Well | 1 |
| Antral excision only | Poor | 2 (additional surgical treatment — 2) |

operations by removal of the antrum alone has not been followed by satisfactory results, since, as previously mentioned, 1 patient died after the operation, while the 2 who survived had poor results. Both required additional operations, and after these both succumbed. The patient treated with a combination of antral excision and transthoracic vagotomy was completely relieved (table 10).

ADDITIONAL SURGICAL TREATMENT

Ten patients who obtained poor results from the first operation for jejunal ulcer in our hospital required further surgical treatment from us or elsewhere. The cases are summarized in table 11. The operations for jejunal ulcer which had failed in the 10 cases were: (*a*) closure of gastroenterostomy opening in 2 cases; (*b*) closure with pyloroplasty in 1 case; (*c*) antral excision in 2 cases; (*d*) Finsterer exclusion in 1 case; (*e*) subtotal gastrectomy in 3 cases, and (*f*) reresection in

1 case. Six of the 10 patients underwent one additional operation, and 4 required two additional procedures. Seven patients subsequently died, 3 of them after these secondary operations and 3 because of a continuation of their disease. The result in 1 case is unclassified, as

TABLE 11.—*Cases in Which Additional Surgical Treatment Was Required*

| Patient, Sex, Age | Course | | Final Result |
|---|---|---|---|
| B. I. | Duodenal ulcer, posterior gastroenterostomy | 1914 | |
| Male | Dismantling of gastroenterostomy mechanism | 7/11/35 | |
| 58 | Anterior gastroenterostomy | 8/ 7/36 | |
| | Died after recurrence | 9/16/36 | Poor |
| W. B. | Duodenal ulcer, posterior gastroenterostomy | 1931 | |
| Male | Dismantling of gastroenterostomy mechanism | 5/16/47 | |
| 54 | Subtotal gastrectomy | 4/27/48 | |
| | Well, with minor side effects | 11/ 6/48 | Well |
| J. J. | Duodenal ulcer, posterior gastroenterostomy | 10/ 7/24 | |
| Male | Dismantling of gastroenterostomy mechanism, with pyloroplasty | 10/14/26 | |
| 28 | Posterior gastroenterostomy (elsewhere) | 1945 | |
| | Transthoracic vagotomy | 8/ 8/46 | |
| | Improved—hypertrophic gastritis | 6/ 1/48 | Improved |
| F. B. | Duodenal ulcer, Finsterer exclusion * | 11/29/41 | |
| Female | Antral excision | 1/ 7/42 | |
| 39 | Total gastrectomy | 6/24/42 | |
| | Died from peritonitis | 6/27/42 | Poor |
| F. Y. | Gastric ulcer, Finsterer exclusion * | 4/ 1/43 | |
| Male | Antral excision | 5/19/44 | |
| 62 | Transverse colostomy for gastrojejunocolic fistula | 4/25/45 | |
| | Died from perforation and peritonitis | 6/23/45 | Poor |
| F. S. | Duodenal ulcer, posterior gastroenterostomy | 1928 | |
| Male | Finsterer exclusion | 2/19/38 | |
| 69 | Resection without antrumectomy | 4/17/41 | |
| | Resection with antrumectomy | 4/ 3/42 | |
| | Died from pancreatic fat necrosis and peritonitis | 4/23/42 | Poor |
| A. B. | Gastric ulcer, Finsterer exclusion * | 8/ 3/27 | |
| Male | Resection without antrumectomy | 9/26/28 | |
| 42 | Closure, acute perforation of jejunal ulcer (elsewhere) | 1937 | |
| | Exclusion of jejunal ulcer | 8/12/38 | |
| | Died from cerebral hemorrhage | 1/ 5/39 | Poor (?) |
| F. B. | Duodenal ulcer, posterior gastroenterostomy * | 7/23/27 | |
| Male | Subtotal gastrectomy | 1/20/30 | |
| 25 | Dismantling of posterior gastroenterostomy mechanism and establishment of anterior gastroenterostomy opening for gastrojejunocolic fistula (elsewhere) | 2/17/41 | |
| | Died | 2/23/41 | Poor |
| G. R. | Duodenal ulcer, posterior gastroenterostomy | 10/10/29 | |
| Male | Subtotal gastrectomy | 1/11/39 | |
| 41 | Repair of gastrojejunocolic fistula (elsewhere) | 2/22/47 | |
| | Anterior gastroenterostomy and vagotomy (elsewhere) | 10/23/47 | |
| | Well | 6/14/48 | Well |
| T. L. | Duodenal ulcer, posterior gastroenterostomy | 1936 | |
| Male | Subtotal gastrectomy | 11/10/44 | |
| 53 | Ileosigmoidostomy for gastrojejunocolic fistula | 5/ 4/46 | |
| | Died from inanition and sepsis | 6/ 5/46 | Poor |

* The original operation was performed in University Hospital.

death from cerebral hemorrhage occurred five months after the last operation, the patient having remained symptom free during that brief period. Of the 3 living patients, 2 have ultimately become well, while 1 is improved. Cure was accomplished in 1 patient, now symptom free, by subtotal gastrectomy after closure of a gastroenterostomy open-

ing had failed and in the other by vagotomy after subtotal gastrectomy had failed. The patient listed as improved was partially relieved by transthoracic vagotomy after three previous operations for ulcer had been unsuccessful. It is believed that his residual symptoms are due to a persistent hypertrophic gastritis, which is demonstrated both by roentgenograms and by gastroscopy.

The 2 patients who were unrelieved by antral excision deserve special comment. In 1 case a Finsterer exclusion operation was performed as the first stage of a classic resection as advocated by McKittrick. A jejunal ulcer developed before the second stage could be undertaken. This was uninfluenced by the antral excision, and five months later the remaining gastric pouch was completely removed. Death occurred because of peritonitis consequent on leakage at the esophagojejunal anastomosis. Microscopic examination of the specimen revealed a large ulcer in the jejunum, with associated carcinoma. Autopsy disclosed a solitary metastasis in the thyroid gland. This case is unique in our experience. The second patient underwent what was considered to be an adequate resection for a gastric ulcer of the lesser curvature, although at that time the surgeon was not certain of the exact level of the lower transection in relation to the pylorus. Symptoms recurred shortly thereafter, and a jejunal ulcer was demonstrated by roentgenologic study. The gastric acidity remained high, and accordingly resection of the upper portion of the duodenum was performed, with the hope of eradicating any remaining antral mucosa. Microscopically, antral tissue was identified. The patient was not relieved by this procedure, a gastrojejunocolic fistula developed and, in spite of a proximal colostomy, death occurred from perforation and peritonitis.

Gastrojejunocolic fistulas subsequently developed in 3 other patients, all following gastric resection for jejunal ulcer. Two of the patients died, 1 after an ileosigmoidostomy and 1 after direct repair. The third patient is now well, after a reparative operation for the fistula and a gastroenterostomy with vagotomy eight months later.

## COMMENT

The prevention of jejunal ulcer consists first in the proper selection of patients with primary gastroduodenal ulcer for surgical treatment and second in the selection of the operative procedure best suited to the individual case. In this clinic gastrojejunostomy has become practically obsolete during the past decade, although recently it has been temporarily revived as an adjunct in certain cases of vagotomy. Vagotomy, either alone or in conjunction with other surgical procedures, has been employed in 64 cases of gastroduodenal ulcer, with the following results: good in 41 (64.1 per cent), fair in 17 (26.5 per cent) and poor in 6

(9.4 per cent). A detailed report of these cases is to be made by Fuller.[24] The employment of vagotomy has been temporarily discontinued until a more thorough evaluation of the results in these cases is made. Subtotal gastrectomy remains the standard procedure for both gastric and duodenal ulcer, since in our hands it has given eminently satisfactory results, with a low operative mortality. When the classic operation is attended by difficulty in the management of the duodenal stump, Bancroft's modification of the Finsterer procedure is usually preferred.

In the definitive treatment of gastrojejunal ulcer following gastroenterostomy, the dismantling operations, with restoration of the gastrointestinal tract to normal, have proved unsatisfactory, and there would now seem to be few if any indications for their use. An adequate gastric resection, on the other hand, probably offers the greatest chance of cure. When recurrence follows a conventional or an exclusion gastrectomy, the status of the original resection should be carefully evaluated. If the antrum remains or if the resection was small, a resection that is adequate as judged by present day standards is indicated. When recurrence is encountered in patients who already have been subjected to an acceptable resection, vagotomy affords a new and promising mode of treatment, whereas formerly for this group of patients only total or nearly complete gastrectomy remained.

24. Fuller, W. J.: Personal communication to the author.

## DISCUSSION

DR. R. L. SANDERS, Memphis, Tenn.: The subject of vagal resection in the treatment of duodenal and gastrojejunal ulcer is one in which I have been deeply interested since I performed my first operation of this type more than three years ago. Our series now comprises about 150 cases. Eight of the vagal resections have been performed for gastrojejunal ulcer, six of the eight more than a year ago. Six of the patients underwent vagal resection alone, while 2 were subjected to gastric resection as well. To the present time, all these patients have had a good or excellent result. In the 2 who required resection of the stomach there was obstruction both at the pylorus and at the stoma. It has been our observation that if obstruction is appreciable, the combined gastrectomy and vagal resection is advisable; if the stomach empties well, however, resection of the nerves alone is sufficient.

Being abdominal surgeons, we have employed the abdominal approach in all our vagal resections. We believe that this approach is preferable to the transthoracic, chiefly because of the fact that one is thus able to visualize the disease process, but also because the patients are more likely to have a comfortable postoperative course. So far as we have been able to determine, the symptomatic and clinical results are as good as those obtained after the use of the transthoracic approach.

# REPEATED MASSIVE INTESTINAL HEMORRHAGE FROM MULTIPLE MALIGNANT TUMORS OF THE JEJUNUM

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THE FOLLOWING case seemed to us to demonstrate some difficulties in making a clinical diagnosis of tumors of the small intestine and exhibited several unusual features of sufficient interest to justify reporting it.

## REPORT OF A CASE

The patient was a white married man, 58 years of age. His family history and past history were noncontributory. He was admitted to the hospital on March 31, 1948, to the medical service of Dr. Don C. Wakeman, with a history of tarry stools for one month and weakness and dizziness on exertion for two weeks. Two days before his admission he had fainted after taking a few steps.

Examination demonstrated no significant findings except pallor. His hemoglobin content was 40 per cent, with 2,490,000 erythrocytes. Other laboratory studies revealed nothing abnormal except that a stool specimen contained blood. The provisional diagnosis was bleeding duodenal or gastric ulcer.

He was treated by ordinary conservative measures, including transfusions. A few days after admission roentgenologic study of the gastrointestinal tract demonstrated a small hiatus hernia with a congenitally short esophagus, but no ulcer or tumor was visualized.

As the tarry stools continued, the idea of an ulceration due to the hiatus hernia was seriously considered, and phrenicectomy was performed on the left side on April 10, with the hope that paralysis of the left side of the diaphragm might relieve pressure on an ulcerated area if it was due to the hernia. There was a temporary cessation of active bleeding, but on April 28 he passed another large tarry stool, with others on following days, and his hemoglobin content decreased to 34 per cent (with 2,269,000 erythrocytes). At this time it seemed that the stomach might not be the source of the hemorrhage after all, since no definite ulceration could be demonstrated, there had been no permanent improvement after the phrenicectomy and the blood had been passed by rectum but never vomited. Exploratory laparotomy was advised, with the provisional diagnosis of a small intestinal lesion as the source of the bleeding.

After preparation, the abdomen was explored on May 9. The lower part of the small intestine and the colon were found filled with blood, but there was none in the upper part of the jejunum. A polypoid lesion 1 cm. in diameter was felt through the wall of the upper region of the jejunum, attached near the mesenteric

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side. The serosa over the lesion showed hemorrhagic stippling. There was no evident involvement of either regional lymph nodes or liver, and no other lesions of the intestinal tract were noted. A segment of jejunum, including the tumor, was resected and the continuity restored by end to end anastomosis.

The postoperative course was unexpectedly smooth, and the patient was dismissed from the hospital on the tenth postoperative day, with his hemoglobin content rising although he had not been given any additional transfusions postoperatively.

Grossly, the lesion was a spherical tumor about 1 cm. in diameter protruding into the lumen of the bowel, apparently located in the submucous layer and attached at the mesenteric border. On the apex of the growth there was an ulcerated area. The microscopic report was "carcinomatous polyp of the small intestine."

The patient made excellent progress for six weeks after his dismissal from the hospital, when he again began to feel badly and to pass large tarry stools. He was readmitted on July 17, and again the only significant finding on examination was extreme pallor. His hemoglobin content on this occasion was 37 per cent, with 2,030,000 erythrocytes.

With symptoms the same as on his previous admission, the possibility of a similar lesion in another part of the intestinal tract was, of course, immediately suggested. In order to eliminate the stomach as a source of the bleeding, aspiration was carried out with an indwelling Levin tube continuously over a period of nearly twenty-four hours at a time when the patient seemingly continued to bleed. No blood, however, was returned from the stomach during any of this time. The prothrombin content of the blood, the bleeding time, the coagulation time and the clot retraction were all normal. With a presumptive diagnosis of another bleeding lesion of the small intestine, he was prepared for operation without further study.

At operation on July 23, a second tumor—identical in gross appearance to the one removed ten weeks previously—was found on the mesenteric side of the jejunum less than 1 inch (2.5 cm.) below the duodenojejunal junction and some 24 to 30 inches (61 to 76 cm.) above the original anastomosis. A careful search throughout the length of the intestinal tract revealed no other lesions. There was no evidence of any metastatic spread, and the site of the previous anastomosis was in good condition. After consideration of the technical difficulties in resecting the intestine at the level involved, the tumor was removed through an incision on the opposite side of the intestine and the opening closed transversely.

The patient again made a good recovery and was dismissed from the hospital on his fourteenth postoperative day, with a hemoglobin level of 81 per cent and an erythrocyte count of 4,330,000.

The microscopic appearance of the two tumors was identical.

Microscopic slides of this tumor, together with clinical abstracts, were submitted to four consultant pathologists in addition to the two who studied the lesion primarily. The variety of opinions expressed demonstrates the doubtful nature of the tumor.

As noted previously, the original diagnosis of the local pathologists was "carcinomatous polyp." The same diagnosis was originally made for the second tumor, but later, after further study, this was changed to "carcinoid."

One of the consultant pathologists likewise made the diagnosis of "carcinoid," with the following comments: "In my opinion, the type of tumor with which you are dealing in this patient is that which has been called carcinoid, the exact nature of which is not known. When carcinoids appear in the appendix, they are

usually benign. However, it has been my experience that when they are seen in the small intestine they are almost universally malignant. This one which you have, I think, is of a malignant type. Rarely they are multiple when seen in the small bowel, and I think you are dealing with multiple lesions rather than with metastasis or implantations."

The second opinion was that "leiomyosarcoma" was present, with comments as follows: "The structure is obviously that of a smooth muscle tumor, and the nuclei show changes that are characteristic of malignant growth. Mitoses are infrequent, but the bizarre appearance, the extreme variation in size and the presence of multiple nuclei point to a relatively malignant tumor. My diagnosis is leiomyosarcoma."

A third diagnosis was "anaplastic metastatic tumor to small intestine," and the consultant commented: "From the microscopic picture it is evident that this represents a highly anaplastic malignant type of tumor. On the basis of the suggestive location within lymphoid tissue, the presence of two lesions at the site of mesenteric attachment and the greater frequence of metastatic tumors in relation to primary tumors of the small intestine, it seems highly probable that this represents a metastatic lesion, even though the history and the physical condition of the patient do not at present substantiate this diagnosis. On the basis of the material submitted it is my opinion that this is most probably a metastatic malignant melanoma of the amelanotic variety. The small intestine is the not infrequent site of metastasis from this type of tumor, even in the absence of any obvious or recognized primary site for the tumor. A second possibility is that this might represent a metastatic leiomyosarcoma."

This same consultant also showed the slides to three of his friends, one of whom agreed with him; the other two placed metastatic leiomyosarcoma as first choice, with metastatic melanoma as a second choice.

The fourth consultant's diagnosis was "metastatic carcinoma, melanoma to be ruled out," and his comments were as follows: "There are several possibilities which should be considered in multiple malignant tumors of the small bowel. Carcinoids are often multiple, usually occur near the ileocecal valve, have a yellowish appearance grossly and microscopically show a characteristic picture. Invariably carcinoids have rather small cells with small nuclei, and mitotic activity is minimal. I do not believe that this could be a carcinoid; they practically never give rise to gross hemorrhage. Leiomyosarcomas can give rise to massive hemorrhage, but when they do they are usually large. They also have spindle-shaped cells, and their microscopic pattern is not like that of this tumor. Plasma cell tumors have been reported as being multiple in the small bowel, but their microscopic recognition is easy, and individual cells resemble plasma cells. Lymphosarcomas do not usually show multiple nodules in the small bowel unless the process is fairly extensive, and this slide does not show any of the characteristics of lymphosarcoma. Metastatic neoplasms, when they involve the small bowel, usually grow in the submucosa. Such neoplasms usually occur when the process is extensive. However, in some instances the primary lesion can be small. This is particularly prone to occur in malignant melanomas. Questionable but not definite melanin pigment is seen. This patient should be questioned carefully about the removal of a previous mole. The time interval between the removal and the appearance of this tumor could be long—even ten years. I believe that other lesions will eventually develop, and the patient will eventually die of this process."

*Follow-Up.*—The patient was seen on Nov. 27, 1948, when he had the appearance of perfect health. He stated that he had not noticed any abnormal

coloration of his stools since returning home early in August. He had gained weight up to his normal of 165 pounds (74.8 Kg.). His hemoglobin level was 85 per cent, with an erythrocyte count of 4,400,000.

He was examined again for any evidence of other primary malignant tumor, and nothing suggestive was found. He does have numerous pigmented moles distributed over his body, but none suggested malignant change, and he does not know of any of them having changed color or enlarging. He has never had any moles removed.

The ultimate prognosis is, of course, guarded at best, in spite of the favorable condition at the present time.

## COMMENT

A discussion of all the tumors of the small intestine is not within the province of this paper, but a brief consideration of the circumstances present may be in order.

Malignant tumors of the small intestine are not rare, but they are certainly uncommon. During a period of thirty-two consecutive years only 108 malignant tumors of the small intestine were encountered at the Mayo Clinic.[1]

Multiplicity is noted in a small percentage of patients having malignant tumors, the number varying from 0.86 to 4.7 per cent (Abell).[2] Slaughter, in a detailed analysis of multiple primary malignant tumors, recorded 1,868 cases for which he could find suitable records. In 749 of these there were multiple lesions in one organ, and of these only four were of the small intestine—three in the ileum, one in the duodenum and none in the jejunum.[3]

The accepted criteria for the diagnosis of multiple primary malignant tumors[4] are not fulfilled by the present case, principally because the two lesions have identical gross and microscopic structure but also because there are difficulties in proving that one lesion was not secondary to the other or that both were not metastatic from some unknown focus.

Bleeding is an expected symptom of tumors of the small intestine, but it is more commonly as occult blood in the stool than as a massive hemorrhage when it is the only presenting symptom. Massive hemorrhages are more likely to be from benign tumors of this locality than

1. Mayo, C. W.: Carcinoma of the Small Intestine, West. J. Surg. **48**:403, 1940.

2. Abell, I.: Multiple Primary Malignant Growths, South. Surgeon **2**:39 (March) 1933.

3. Slaughter, D. P.: The Multiplicity of Origin of Malignant Tumors, Internat. Abstr. Surg. **79**:89 (Aug.) 1944.

4. Eisenstaedt, J. S.: Multiple Primary Malignant Tumors, J. A. M. A. **110**: 2056 (June 18) 1938. Hurt, H. H., and Broders, A. C.: Multiple Primary Malignant Neoplasms, J. Lab. & Clin. Med. **18**:765 (May) 1933.

from malignant ones.[5] If massive hemorrhage from one malignant lesion in this region is uncommon, then the same thing from a second lesion must be even less frequent.

This case demonstrates some of the difficulties of making a diagnosis of tumors of the small intestine. To make the diagnosis one must first think of the possibility of such a lesion; symptoms are often vague and in most cases are not distinctive of the level of the tumor. Specific symptoms consist primarily of obstruction or bleeding.[6] Roentgenologic studies of the small intestine may identify a tumor of this type, but special technics are usually required.[7] Some are palpable when first seen, but they are apt to be inoperable at that time. Siper and Wood stated in a summary that the "clinical diagnosis of any tumor of the small bowel is usually quite a difficult procedure and as often made on the basis of a systematic elimination of the possibilities that the lesion is in some other organ."[8] Bleeding which produces melena without hematemesis is more likely to have its origin below the ligament of Treitz than above it (Smith, Good and Gray),[9] and exploratory laparotomy has a recognized role in the diagnosis of lesions of this type when there is bleeding from the intestinal tract for which no source can be found in the esophagus, stomach, duodenum or colon.

The only treatment of curative value is surgical extirpation, and the prognosis is bad in cases of malignant growths. As Fraser described it, "bad in the immediate future because so many are obstructed when first seen, and bad in the distant future because of the frequency of metastases."[10] McDougal estimated the incidence of five year sur-

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8. Siper, J., and Wood, E. C.: Metastatic Melanocarcinoma Involving the Small Intestine and Brain, New England J. Med. **236**:824 (May 29) 1947.

9. Smith, L. A.; Good, C. A., and Gray, H. K.: Tumor of the Small Intestine as the Cause of Recurrent Melena: Report of Two Cases, Proc. Staff Meet., Mayo Clin. **19**:117 (March 8) 1944.

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vivals at 5 per cent—a striking contrast to the approximate 50 per cent of patients having carcinoma of the colon who survive five years.[11]

In conclusion, a report is made of a patient who had repeated massive gastrointestinal hemorrhages, ultimately proved to have their origin in two distinct malignant tumors of the jejunum, which were resected with an intervening interval of ten weeks. No claim is made that the lesions were definitely independent primary tumors, although some are of the opinion that they were. The pathologic diagnosis has been that the lesions were malignant without question, though the type is not definite, a carcinoid, a primary leiomyosarcoma, a metastatic melanoma and a metastatic leiomyosarcoma having been considered. Symptoms and findings which may suggest the diagnosis of tumors of the small intestine are briefly discussed. In questionable cases exploratory operation may be the only means of recognition.

11. McDougal, W. J.: Carcinoma of Small Intestine, Am. J. Surg. **66**:119 (Oct.) 1944.

## DISCUSSION

DR. JAMES A. JACKSON, Madison, Wis.: I should like to reiterate the fact that in severe intestinal hemorrhage of unexplained origin the value of early roentgen examination of the small intestine is of utmost importance, and I wish to review briefly a case that we encountered approximately six months ago.

A woman came to the clinic evidently having severe intestinal hemorrhages. No definite diagnosis of the source of this hemorrhage was made until she had made two or three visits. Her hemoglobin concentration was low; the red blood cell count was about 1,000,000. Finally, a roentgen examination of the small intestine demonstrated an involvement of the jejunum by a tumor mass.

I operated on this patient and found a carcinoma of the jejunum which had involved most of the small intestine by direct extension between adjacent loops. There was no apparent involvement of the mesentery, and in attempting a resection I found it necessary to remove practically the whole jejunum and ileum. In fact, if the anatomists are correct in saying there are 20 feet (660.4 cm.) of small intestine, I removed approximately 18½ feet (564 cm.). A pathologic examination demonstrated no lymphatic involvement in spite of a careful study. This phenomenon seemed highly improbable considering the extent of the lesion.

It has been approximately six months since the operation, and the patient has made an uneventful recovery so far. She has about 8 inches (20 cm.) of jejunum and ileum left, that is, about 4 inches (10 cm.) of the jejunum and 4 inches of the ileum. We have been able to maintain her normal weight and with dietary control, and she has only two semisoft stools a day.

A review of the literature up to this time has revealed no case in which there has been such a massive resection of approximately 18½ feet of small intestine, with the patient surviving and apparently doing well after six months. If the patient is still alive next year, I hope to make a full report on the case at our meeting.

DR. RALPH H. LOE, Seattle, Wash.: This interesting paper illustrates the difficulty of the diagnosis of lesions of the small bowel. Several years ago Dr. Abbott pointed out that the Miller-Abbott tube was a valuable adjunct in the localization of lesions of the small bowel. In 1942 I had occasion to prove its

value in a patient who had had four massive hemorrhages of the upper part of the gastrointestinal tract. Repeated roentgen examinations and a gastroscopic examination had revealed no abnormalities. On her fourth admission to the hospital a Miller-Abbott tube was introduced into the duodenum, and when the tip had progressed to a point 1 foot (30.48 cm.) beyond the pylorus bright red blood was obtained by continuous suction. Surgical exploration revealed a small leiomyoma just beyond the ligament of Treitz, which I feel certain would have been missed without a rough idea of the localization of the source of difficulty.

Dr. H. E. Snyder, Winfield, Kan.: I have enjoyed this presentation very much. Malignant tumors of the small bowel are certainly infrequent. The variety of reports received on the tissue from these two rather innocuous-appearing lesions is indeed interesting.

When I was an intern at Pennsylvania Hospital in 1929, a man was admitted to the surgical service who had been operated on eighteen years previously by Francis T. Stewart. At that time a malignant lesion of the small bowel and jejunum had been removed, and the pathologist's diagnosis had been spindle cell sarcoma of the intestine. It was of such rarity that the specimen was put in the museum of pathology.

After an examination of this patient and the finding of a retroperitoneal mass, and biopsy thereof, we were able to compare sections from the lesion which appeared in these retroperitoneal lymph nodes eighteen years after the original tumor. Both appeared identical. That man had lived eighteen years without symptoms.

I am sure we can appreciate Dr. Mills's and Dr. Clark's difficulty in offering some sort of prognosis in this case.

Dr. O. R. Clark, Topeka, Kan.: I have little to add except to thank the three discussers for their comments on the paper and the presentation of these other cases.

I might mention that in the cases of tumor of the small intestine which are found in the literature approximately one half of the tumors were found only incidentally either at autopsy or at operation, having produced no symptoms which would call attention to their presence.

# POLYPOSIS OF THE COLON

E. LEE STROHL, M.D.
AND
GUY V. PONTIUS, M.D.
CHICAGO

DIFFUSE polypoid disease of the colon, or multiple polyposis by common usage, occurs infrequently. It has been recognized since 1863,[1] when the pathologic characteristics were described by Virchow.

The heredofamilial character,[2] the diffuse involvement of the colon,[3] and the tendency to malignant change in early life make the disease significant.[4] The cases may be divided into two major types, as suggested by Erdmann and Morris:

(1) Adult or acquired type, usually occurring in middle or late adult life and characterized by a prolonged history of colitis. There are fewer polyps throughout the colon in this type.

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From the Department of Surgery, St. Luke's Hospital.

Read at the Fifty-Sixth Annual Meeting of the Western Surgical Association, St. Louis, Dec. 3, 1948.

1. Virchow, R.: Die krankhaften Geschwülste.: Dreissig Vorlesungen, gehalten während des Wintersemesters 1862-1863 an der Universität zu Berlin, Berlin, A. Hirschwald, 1863, p. 242.

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3. Cripps, W. H.: Two Cases of Disseminated Polyposis of the Rectum, Tr. Path. Soc. London **33**:158-165, 1882. Falk, V. S.: Familial Polyposis of the Colon, Arch. Surg. **45**:123-129 (July) 1942. Friedell, M. J., and Wakefield, E. G.: Familial Polyposis of the Colon, J. A. M. A. **121**:830 (March 13) 1943. Hoxworth, P. I., and Slaughter, D. P.: Polyposis (Adenomatosis) of the Colon, Surgery **24**:188-211 (Aug.) 1948. Hullsiek, H. E.: Multiple Polyposis of the Colon, Surg., Gynec. & Obst. **47**:346-356 (Sept.) 1928. Lahey, F. H.: Polyposis of the Colon, Lahey Clin. Bull. **4**:130-133 (July) 1945. Lockhart-Mummery, J. P., and Dukes, C. E.: Familial Adenomatosis of Colon and Rectum, Lancet **2**:586-589 (Sept. 9) 1939.

4. (*a*) Erdmann, J. F., and Morris, J. H.: Polyposis of the Colon: A Survey of the Subject, Surg., Gynec. & Obst. **49**:460-468 (April) 1925. (*b*) Jackman, R. T.: Relationship of Polyps of Colon to Cancer, Proc. Staff Meet., Mayo Clin. **16**:11-12 (Jan. 2) 1941. (*c*) McLaughlin, C. W., Jr.: Diffuse Polypo-

(2) Adolescent (congenital disseminated) type occurring in youth or young adult life. A history of a similar condition may be obtained from other members of the family. The symptoms are recurrent episodes of abdominal discomfort, associated with cramps and bloody diarrhea. The colon is usually diffusely studded with polyps from the cecum to the anus. The polyps are not present in the susceptible persons at birth but appear in childhood or near the age of puberty. This factor results in much difficulty in classification. It therefore necessitates a careful elicitation of the history and thorough examination of other members of the family to establish a diagnosis of heredofamilial polyposis.

Irrespective of the basis on which polyposis develops, the incidence of carcinoma is unusually high. Scarborough stated that "probably no benign process has a higher incidence of malignant degeneration than polyposis." In his collected series the incidence of carcinoma was found to be above 50 per cent. Hullsiek reported that 34.6 per cent of his collected series presented evidence of malignant degeneration. We think that carcinoma of the large bowel or rectum will develop in 100 per cent of patients with polyposis of the colon if they survive a sufficient number of years. Persons with the heredofamilial form of the disease tend to exhibit malignant degeneration at a younger age than those with the acquired form of the disease.[5]

Other complications of the disease are anemia and wasting, secondary to the prolonged diarrhea and loss of blood.[6] Partial or complete

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sis of the Large Intestine, Am. J. Surg. **62**:258-266 (Nov.) 1943. (*d*) Miller, R. H., and Sweet, R. H.: Multiple Polyposis of the Colon: Familial Disease, Ann. Surg. **105**:511-515 (April) 1937. (*e*) Pfeiffer, D. B., and Patterson, F. M. S.: Congenital or Hereditary Polyposis of the Colon, ibid. **122**:606-624 (Oct.) 1945. (*f*) Pugh, H. L., and Nesselrod, J. P.: Multiple Polypoid Disease of the Colon and Rectum, ibid. **121**:88-99 (Jan.) 1945. (*g*) Rankin, F. W.: Colectomy for Adenomatosis and Pseudopolyposis, ibid. **102**:707-723 (Oct.) 1935; (*h*) Multiple Polyps of the Colon, South. M. J. **27**:574-578 (July) 1934. (*i*) Wesson, H. R., and Bargen, J. A.: Classification of Polyps of the Large Intestine, Proc. Staff Meet., Mayo Clin. **9**:789-794 (Dec. 26) 1934.

5. Coffey, R. J., and Bargen, J. A.: Intestinal Polyps: Pathogenesis and Relation to Malignancy, Surg., Gynec. & Obst. **69**:136-145 (Aug.) 1938. Saint, J. H.: Polypi of the Intestine, with Special Reference to the Adenomata, Brit. J. Surg. **15**:99-119 (July) 1927-28.

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*(Footnote continued on next page)*

obstruction incident to enlargement of the polyps has been observed. Intussusception of various segments of the colon may be expected.

It is the purpose of this paper to present 3 cases of polyposis of the colon occurring in young adults, with cancerous degeneration in the involved colon.

CASE 1.—*History.*—A. W. T., a 46 year old white man, entered St. Luke's Hospital on Sept. 17, 1940. He complained of abdominal distention following meals and frequent stools, with occasional bloody diarrhea, of four years' duration. He lost 20 pounds (9.1 Kg.) in weight during the preceding nine months. Although the symptoms extended over a four year period, medical consultation was sought only after friends had commented on his progressive loss in weight.

The past history was noncontributory. The family history revealed that his father died at the age of 72 of recurrent cancer of the colon. His maternal grandfather died at 72 of cancer of the stomach. One sister died at 25 after perforation of a cancer of the colon. His mother was living and well at 81 years.

*Proctoscopic Examination.*—Many polyps were seen, both sessile and pedunculated, with a fungating annular cancer of the rectum at 14 cm. A specimen of the tumor was removed for microscopic study.

*Roentgenologic Examination.*—Barium enema revealed a filling defect of the rectosigmoid, with questionable polyps of the entire colon above the lesion.

After a few days' preparation with administration of sulfaguanidine, low residue diet, transfusions and vitamin therapy, the patient was operated on on Oct. 4, 1940.

*Surgical Procedure.*—A low midline incision was made. Exploration revealed the entire descending colon, transverse colon, ascending colon and cecum filled with polyps of varying sizes. Just below the peritoneal reflection was an ulcerated carcinoma. Many regional lymph nodes were palpable. The liver was normal on palpation. The operation was completed as an abdominoperineal resection. The postoperative course was uneventful, the patient being discharged on the eighteenth postoperative day.

*Pathologic Changes.*—The excised surgical specimen comprised 36 cm. of colon and rectum (fig. 1). The entire mucosa was studded with polyps, ranging in size from 3 mm. to as much as 3 by 1.7 by 1.3 cm. Most of the polyps were pedunculated, a few being sessile. Fourteen centimeters above the anal ring was a lesion 7.5 by 5.5 cm., with a raised margin and ulcerated base. Microscopic study revealed a carcinoma. Immediately above the carcinoma were larger pedunculated polyps, one measuring 6 by 5 cm. This large polyp was interpreted as papillary carcinoma. No metastatic cancer was found in the lymph nodes (fig. 2).

*Subsequent Course.*—In May 1948 the patient reentered St. Luke's Hospital for a routine examination. During the seven and one-half year interval, he had been in excellent health. On one occasion there had been slight bleeding from the colostomy opening for a few days.

Examination of the stoma with a proctoscope revealed the colon studded with polyps of varying sizes.

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of the Colon: Familial Character and Malignant Tendency, J. A. M. A. **107**:1871-1876 (Dec. 5) 1936. Wilensky, A. O.: Total Colectomy for Polyposis of the Colon with Carcinomatous Degeneration, Surgery **17**:630-634 (April) 1945. Miller and Sweet.[4d] Pfeiffer and Patterson.[4e] Wesson and Bargen.[4f]

Roentgenologic examination revealed polyps throughout the remainder of the colon visualized. Roentgen studies of the chest showed no abnormalities. The blood and urine were normal.

*Comment.*—The patient has refused further surgical intervention for complete removal of the involved colon. He realizes the hazard he is taking. We think that his polyposis may be the congenital disseminated type, in view of the occurrence of carcinoma of the colon in his father at the age of 72 and in one sister at the age of 25. It is possible that this is an acquired type; however,

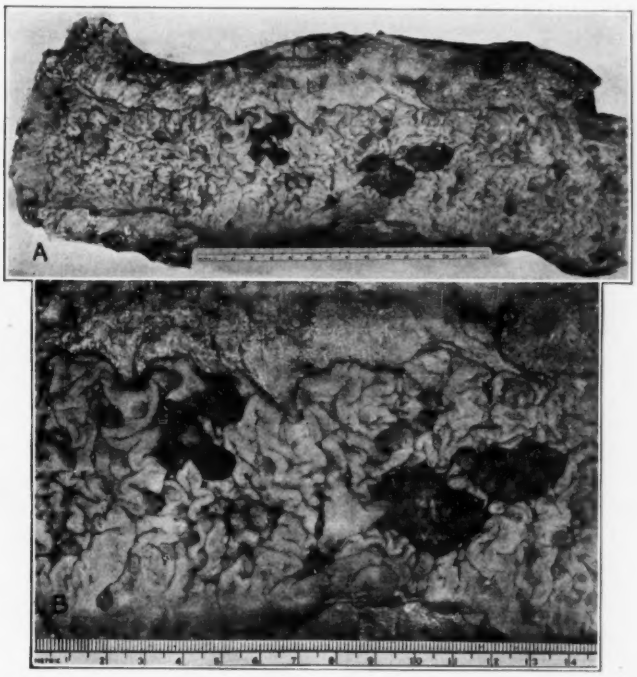


Fig. 1.—*A*, the surgical specimen showing many polyps and the two large polyps which caused the filling defects seen on roentgen examination. *B*, high power magnification showing many polyps with areas of normal mucosa between them. The cancer was demonstrated by proctoscopic examination and by the filling defects on roentgenologic examination.

the history does not bear out long-standing disease of the colon. The appearance of symptoms at the age of 42 is late for the congenital disseminated type.

Case 2.—*History.*—S. R., a 29 year old unmarried woman, entered St. Luke's Hospital on March 11, 1946. She complained of vomiting and cramping mid-abdominal pain of twelve hours' duration. The pain was intermittent in character, occurring at three to five minute intervals. One year previously she had been hospitalized elsewhere for a similar type of distress. She had been discharged

after roentgen studies of the stomach and gallbladder, with a diagnosis of "nervous stomach." During the interval she had been in excellent health.

The family history revealed that her father died at the age of 42 of cancer of the colon and one sister died at 23 of a tumor of the large bowel. A second sister died at 28 of ulcerative colitis. A third sister died at 20 after appendectomy, and after the operation the surgeon told the family that she had had a malignant growth of the large bowel.

*Laboratory Studies.*—Laboratory studies revealed a red blood cell count of 3,880,000 per cubic millimeter. The hemoglobin content was 7.3 Gm. per hundred cubic centimeters. The leukocytes numbered 7,300 per cubic millimeter.

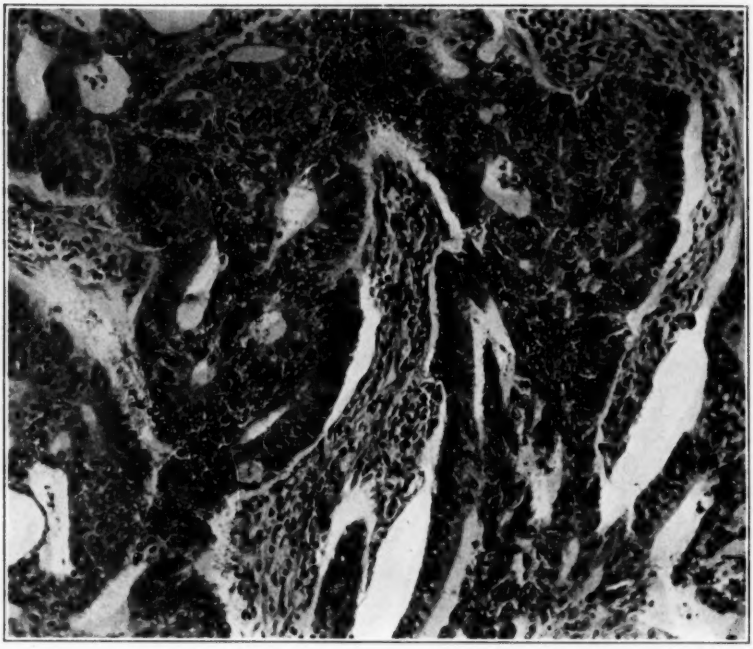

Fig. 2.—Tissue removed from the cancer. The epithelial masses have odd tubule-like lumens. The cells are in several layers, and among them are many in mitosis.

The differential count was normal. The sedimentation rate was 31 mm. in one hour. Examination of the stools revealed occult blood (4 plus). No organisms of the typhoid, paratyphoid or dysentery groups were identified. Chemical examination of the blood and studies of the urine revealed no abnormalities.

*Proctoscopic Examination.*—Many polyps were seen, both sessile and pedunculated, throughout the entire 22 cm. visualized.

*Roentgenologic Examination.*—A thin barium enema revealed a long filling defect, measuring 12 to 14 cm., of the midtransverse colon, extending to the hepatic

flexure. The patient was prepared for surgical treatment with suction decompression, blood transfusions, vitamin therapy and administration of parenteral fluids.

*Surgical Procedure.*—On March 21, 1946, a transverse incision in the upper abdominal region was made. The liver was normal grossly. There were many soft, enlarged lymph nodes in the transverse mesocolon. A constricting lesion was found in the transverse colon. The entire colon, proximal to the lesion, was filled with large and small polyps. A side to side ileotransverse colostomy was made. The cecum, ascending colon and much of the transverse colon were resected. The postoperative course was uneventful, and the patient was discharged from the hospital on the sixteenth postoperative day.

*Pathologic Changes.*—The surgical specimen consisted of 15 cm. of ileum, the cecum, ascending colon and transverse colon, a total of 40 cm. of large bowel.

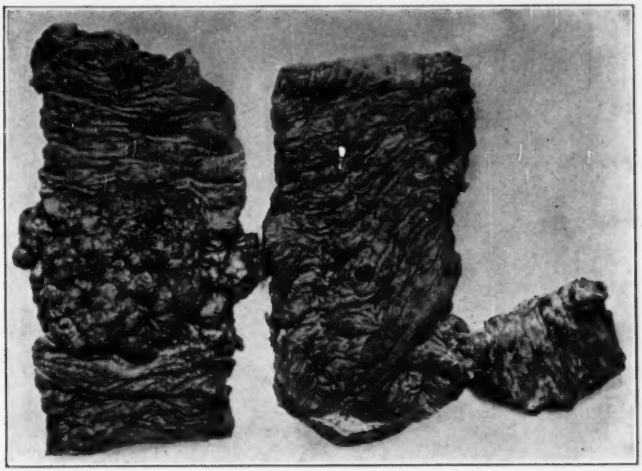

Fig. 3.—Surgical specimen showing the cancer. Both sessile and pedunculated polyps are seen.

Scattered polyps, ranging in size from 4 mm. to 2 cm., both sessile and pedunculated, were found. An annular carcinoma was found in the transverse colon extending 9 cm. longitudinally (fig. 3). Microscopic examination revealed glandular and papillary carcinoma (fig. 4). The regional lymph nodes contained metastatic carcinoma.

*Subsequent Course.*—The patient returned one year later for examination. She was in excellent health and had no complaints.

*Laboratory Studies.*—The blood counts, the chemical composition of the blood, the stools and the urine were normal. A roentgenogram of the chest was normal. Proctoscopic examination revealed many polyps, sessile and pedunculated, throughout the entire 25 cm. visualized. A thin barium enema revealed many polyps in the remaining colon.

*Comment.*—We think that this patient had adolescent or congenital disseminated polyposis, because her father died at 42 of cancer of the colon, one sister died at 23 from a malignant process of the large bowel, a second sister died at 28 of ulcerative colitis and a third sister died at 20 after appendectomy, at which time it was discovered she had a malignant growth of the large bowel.

Further surgical intervention should be carried out; however, this recommendation has been refused repeatedly.

Case 3.—*History.*—R. R. H., a white housewife aged 37, entered St. Luke's Hospital on April 30, 1941. She complained of intermittent diarrhea and bloody stools of seven years' duration. Previous to her admission to the hospital, Amoeba histolytica was found in the stools, and treatment had been instituted elsewhere.

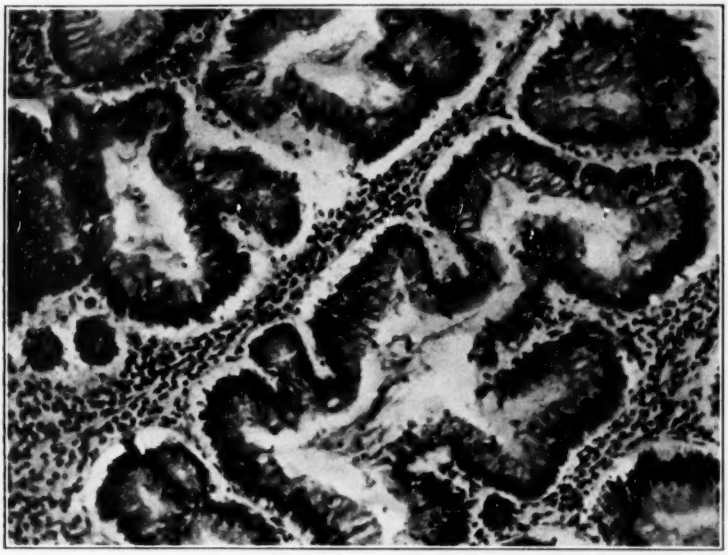

Fig. 4.—Mucoid degeneration of the cancer cells. Atypical glandular tissues with supporting stroma.

Recent examination of stools revealed no amebas, although diarrhea and bloody stools persisted. The family history was noncontributory. She was the mother of three healthy children, aged 8, 5, and 2 respectively.

*Physical Examination and Preoperative Studies.*—The physical examination revealed a healthy woman. The temperature, pulse and respirations were normal. The abdomen was normal. Pelvic and rectal examination showed no abnormalities. Laboratry studies revealed 3,890,000 red blood cells per cubic millimeter. The leukocytes numbered 5,650 per cubic millimeter. The hemoglobin content was 10.5 Gm. per hundred cubic centimeters. The sedimentation rate was 8 mm. in one hour. Chemical studies of the blood revealed no abnormalities. A roentgenogram of the chest was normal.

Examination of the stools revealed a 3 plus reaction to the benzidine test for blood, with no amebas or cysts in the liquid stools.

*Proctoscopic Examination.*—The rectum was studded with polyps varying in size from 3 mm. to 1 cm., some being sessile and others attached by narrow pedicles. At 14 cm. there was a large ulcerated lesion surrounded by polyps, growing in an annular fashion. The instrument was not passed beyond the lesions. A specimen of the tumor removed at the time of the examination revealed cancer.

*Roentgenologic Examination.*—A thin barium enema revealed a constricting annular lesion of the rectosigmoid, interpreted as a malignant growth, with many

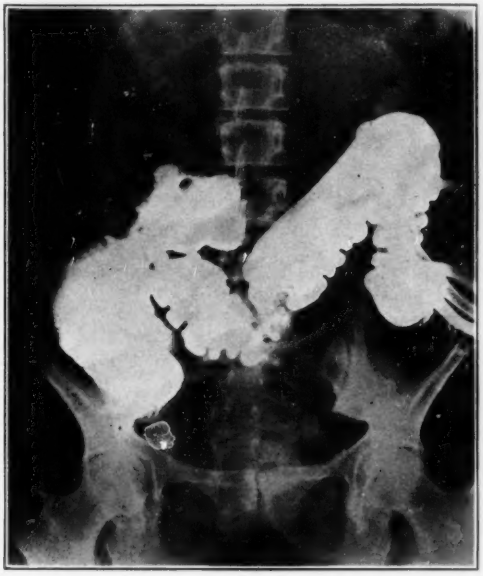

Fig. 5.—Barium enema through colostomy opening revealing an annular constricting lesion of the transverse colon.

polyps of the entire colon. After a few days' preparation with sulfaguanidine, a low residue diet, transfusions and vitamin therapy, the patient was operated on May 15, 1941.

*Surgical Procedure.*—A low midline incision was made. Palpation revealed polyposis of the entire colon, with an annular lesion at the peritoneal reflection. The liver was normal grossly. Many soft lymph nodes were palpable in the mesocolon. The operation was completed as an abdominoperineal resection. Sterilization was accomplished at the time of operation by ligation of the fallopian tubes. During the convalescence, the colostomy opening was examined through a cystoscope, and many polyps were visualized. The convalescence was uneventful, and she was discharged eighteen days after the operation.

*Pathologic Changes.*—The excised surgical specimen comprised 39 cm. of colon and rectum. The entire mucosa was studded with polyps ranging in size from 3 mm. to 3.5 cm. Some were attached by narrow pedicles, and others were sessile. The longest pedicle was 5 cm. Twelve centimeters above the anal orifice was a constricting annular lesion. The mucosa above and below the lesion contained numerous polyps. Microscopic examination revealed the constricting lesion to be carcinoma. All the polyps were benign. Metastatic cancer was found in the regional lymph nodes.

*Subsequent Course.*—The patient was in good health until January 1944, when intermittent pain, with obstructive features, developed. At that time she was readmitted to St. Luke's Hospital. Studies of the remaining part of the colon revealed an annular constricting lesion with polyps (fig. 5). A roentgenologic

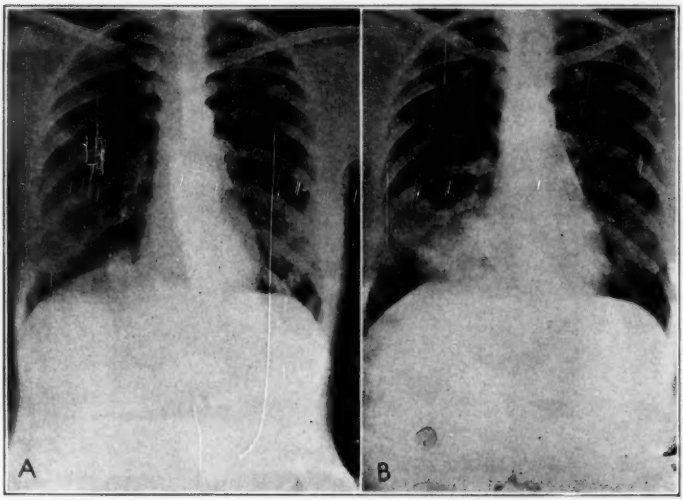


Fig. 6.—*A*, small lesion above the diaphragm interpreted as the residues of recent pneumonia. The possibility of this area being metastatic was considered. *B*, nodular, rounded shadow, clearly demarcated, in the right cardiophrenic angle, which has the appearance of a solid tumor mass.

examination of the chest at that time revealed a small lesion of the right side, interpreted as the residue of pneumonia (fig. 6 *A*). On Jan. 26, 1944, two years and nine months after the abdominoperineal resection, exploration was carried out through a tranvserse incision in the upper abdominal area and the remaining part of the colon resected. Ileostomy was performed in the right lower quadrant. Lymph nodes of generous size were encountered. The postoperative course was uneventful, and the patient was discharged on the fourteenth day.

*Pathologic Changes.*—The excised surgical specimen comprised 60 cm. of colon and 8 cm. of ileum. Thirty-eight centimeters proximal to the colostomy opening, in the transverse colon, was an annular cancer involving the entire wall. Proximal and distal to the lesion, the mucosa was studded with polyps, ranging

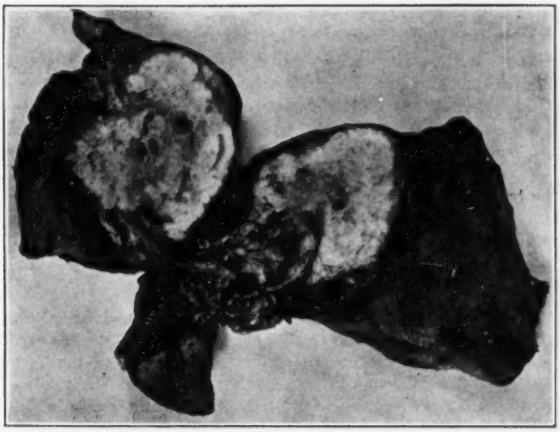

Fig. 7.—Metastatic cancer of the lung. The primary lesion was a cancer of the colon.

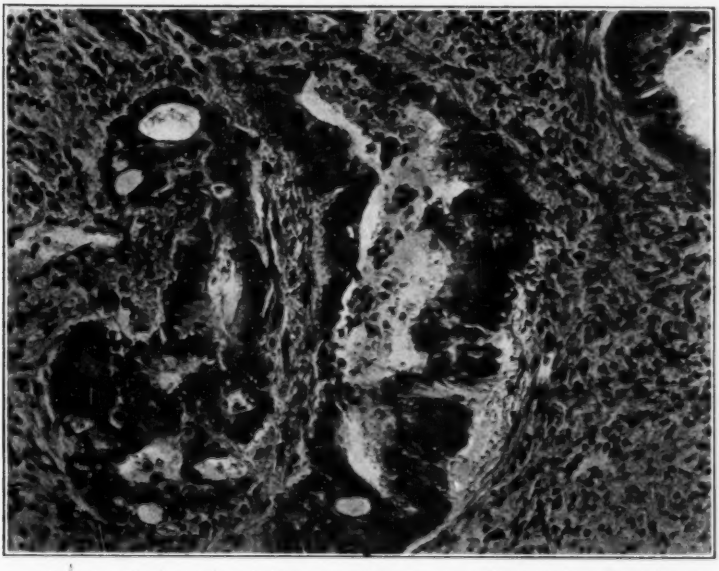

Fig. 8.—The structure of this tumor tissue corresponds to that of a carcinoma of the colon. The cells lining the gland structures are columnar in several layers.

in size from 1 to 5 mm., being both sessile and pedunculated. Regional lymph nodes contained metastatic cancer.

*Subsequent Course.*—The patient was in good health until November 1945, twenty-three months after the second resection of the colon for cancer. A roentgenogram of the chest revealed a nodular, rounded shadow, clearly demarcated in the right cardiophrenic angle which had the appearance of a solid tumor (fig. 6*B*). This mass had been interpreted as the residue of pneumonia twenty-three months previously. Bronchoscopic examination revealed obstruction of the lower part of the bronchus on the right, with soft granular tissue present. Microscopic examination of the tissue revealed metastatic carcinoma, the pathologist reporting that the primary lesion arose in the colon.

*Surgical Procedure.*—On Nov. 2, 1945, thoracotomy was done on the right side and excision of the lower lobe on the right performed.

*Pathologic Changes.*—The surgical specimen revealed glandular metastatic carcinoma of the lung, with metastasis to lymph nodes (fig. 7). The structure of the tumor tissue corresponded to the primary carcinoma of the rectosigmoid (fig. 8).

The postoperative course was uneventful. The patient was discharged, and she carried out an active life for six months. After that time two firm nodules of the scalp and cranium developed and grew rapidly in size. A few days later dysarthria, drooping of the left side of the lower lip and loss of memory for recent events developed. Roentgen ray therapy was suggested; however, the patient died in her home on March 10, 1946, fifty-eight months after the abdominoperineal resection for cancer of the rectosigmoid, with metastatic involvement, and twelve years after the onset of symptoms of diarrhea and bloody stools.

*Comment.*—We think that this patient had congenital disseminated polyposis, although no other members of the family showed evidence of the disease. We are uncertain whether the intrathoracic metastasis originated from the rectosigmoid cancer or from the cancer of the transverse colon. In each instance of resection of the colon, regional lymph nodes were involved with metastatic cancer.

## DISCUSSION

DR. GUY V. PONTIUS, Chicago: The treatment of heredofamilial polyposis is complete colectomy. This should be done because of the strong carcinogenic factor in this disease. Most patients with this type of polyposis will die of carcinoma in the second or third decade, if untreated. It is unusual for such a patient to live beyond the fifth decade without the development of carcinoma of the colon.

Before subjecting a patient to such a radical procedure as colectomy, every caution should be exercised to distinguish this condition from that of multiple, isolated polyps. For such a lesion the removal of the individual tumors is adequate treatment. The carcinogenic factor, while present, is not nearly as strong. In contradistinction to multiple isolated polyps, in heredofamilial polyposis the entire colon, or a large segment of it, is the seat of many polyps varying from a minute size to several centimeters in diameter, both pedunculated and sessile in form. In gross appearance such polyps resemble shag carpeting. A careful elicitation of the history and investigation of blood relatives, will help establish the genetic factor.

The plan of operative procedure will depend on two factors: (1) the condition of the patient, and (2) whether carcinomatous degeneration has occurred. With no evidence of carcinoma, and if the patient is in good condition, ileostomy and complete colectomy may be accomplished in two stages. If for any reason consid-

erable time must elapse between stages, it is well to plan colectomy on the left side first. The vulnerability to malignant degeneration of the left side of the colon versus the right side is comparable to that in carcinoma of the normal colon.

After carcinoma has developed, the plan of procedure is the same as for carcinoma in the normal colon. The advisability and the time for completion of the colectomy are difficult problems. If one is in doubt as to whether the carcinoma has been completely eradicated and if the patient is symptom free it is debatable whether colectomy should be completed. It would seem unreasonable to subject a patient to total colectomy only to have him die of metastatic cancer from the primary lesion. The risk of the development of another carcinoma in the remaining segment must be intelligently weighed.

When these points were discussed with our 3 patients, each elected to wait a three year period unless symptoms developed signifying malignant degeneration in the remaining part of the colon. All submitted to periodic observation.

Ravitch in a recent publication has reported 2 cases of anal ileostomy with preservation of the sphincters and good functional results. The operation in these cases was done for chronic ulcerative colitis. If this procedure proves satisfactory, it should lessen the reluctance of patients to accept complete colectomy.

Dr. Robert W. Bartlett, St. Louis: I agree with the essayists, the Lahey group and others that in resection in these cases the rectum should in most instances be included, and subsequently a permanent ileostomy performed. To eliminate the undesirable features of the permanent ileostomy we have adopted the method of exteriorization of 5 or 6 inches (12.7 to 15 cm.) of the distal end of the ileum, with immediate split thickness skin graft of it, as described by others, and we find this method most satisfactory.

To demonstrate this point, a young man has come some 200 miles today for your inspection. He is a 23 year old veteran who had bleeding off and on for the past four years. He came to us last summer with the complaint of an increased amount of bleeding for three weeks and considerable loss of weight. Last June his operative procedures were started.

(Slide) It was determined, of course, that he had multiple polyposis, and we did first of all a combined abdominoperineal excision and found in the midsigmoid, where the pointer indicates, an obvious carcinoma the size of a quarter. It had not been picked up on roentgenograms because it was too small. There was no evidence of metastasis.

(Slide) This is a low power photomicrograph of the carcinoma. We sent the boy home for a month and then brought him back to complete the colectomy as a second stage operation, and that was carried out the middle of last August.

(Slide) I received this slide just yesterday, and the specimen will have to be rephotographed. It is not a satisfactory picture. The polyps extended to the proximal transverse colon, and in the descending colon a second polyp had undergone malignant degeneration. This patient had two accidentally found early carcinomas.

(Slide) This shows a malignant polyp.

(Slide) This is the high power magnification showing that the growth is clearly malignant. At the time of the second operation we did exteriorize the distal end of the ileum and skin grafted it, and the patient made a satisfactory recovery.

(Slide) This is the skin-grafted organ, which has all the appearances of a penis, and is located in the right lower quadrant.

(Slide) This is a lateral view of the ileostomy. The first stage of the resection was through a left rectus incision, and the second stage was through a transverse incision.

(Slide) This young man wears a Koenig-Rutson air-tight bag. He has five or six semisoft stools each day and goes about his normal life. He is driving to Peoria today to the Hiram Walker Distillery to start work as a guard there. He does not get up at night, and there is no odor. He tells me he was out hunting last week. I think this probably solves the problem of permanent ileostomy.

(Demonstration) In order to remove this air-tight bag he has to use a little ether to melt the cement on his skin. If anyone is interested in seeing the skin-grafted, exteriorized ileostomy opening, I will have the young man at the back of the room for observation after the last paper is presented this morning.

Dr. John H. Woolsey, Woodland, Calif.: I should like to discuss two points in regard to the paper just presented. I noticed that in the summary the authors said they would show the typical roentgenologic findings. I did not see them shown, nor did I hear them discussed, and I hesitate to speak about it because I think all of us probably are familiar with the so-called contrast enema.

We have found that in the confirmation of the diagnosis and proof of this the so-called contrast enema demonstrates it practically 100 per cent. First the patient is prepared. It is most important to be certain that all fecal material is absent. Then the barium enema is given and the patient is allowed to have a bowel evacuation, and then inflation of the bowel is done. Further roentgenograms are then taken, which show the polyps clearly standing out as stalactites. It is really the final proof.

One of the authors spoke about the length of time required and the seriousness of a colectomy. In handling such a lesion we perform the operation as Dr. Henry Cave has suggested it be performed for ulcerative colitis, namely, by the use of the Miller-Abbott tube. If it is passed into the small intestine and this intestine completely emptied, it makes the operation much easier, and the total procedure can be done quickly, in about one third of the time, and with far less shock to the patient.

Dr. C. J. Hunt, Kansas City, Mo.: I am very much interested in this subject of polyposis of the colon, because I am sure that polyps of the colon are a good deal like gallstones—they are not innocent. They lead to ultimate complications, and in this instance they led to a malignant process.

Too often when we make a diagnosis of cancer of the rectum or colon we think the problem of the study of the bowel is completed. We proceed to remove the lesion without making further investigation of the colon to see if there is another cancer or if there are associated polyps. It is wise to find out whether there are polyps that are associated with the malignant lesion and, if there are, whether they are in such a position that, by extending the field of operation, the malignant lesion along with the polyps can be removed.

Therefore, there are two avenues by which to study lesions of the colon and the rectum. Roentgenograms do not reveal accurately malignant lesions or polypoid lesions of the rectum and the rectosigmoid and that portion of the bowel below the pelvic peritoneum or below the lower part of the sigmoid. It is important to study these lesions by proctoscopic and sigmoidoscopic examination. One can then get a biopsy of the polyp and can determine its true cytologic structure.

In order to study the lesions above this region, one has to use roentgenologic investigation. A barium enema gives a great deal of information concerning the colon; it will tell you whether there is a narrowing constricting area of the colon, whether there is an obstruction or whether there is gross deformity of the colon. However, the barium enema itself will cover up the mucosal pattern and you cannot tell a thing about it. You have to do as Dr. Woolsey has suggested, that is, study the entire colon above this area by barium enema and by contrast enema after the opaque medium has been evacuated. Then you can see the opaque medium hanging onto these polypoid projections in the colon and can make a diagnosis of them.

I think we should all establish the principle of studying the entire colon when we are confronted with a malignant lesion of the rectum and of the colon.

(Slide) This slide shows extensive polyposis of the colon in a woman of middle age. This patient refused operation at the time, but later was operated on elsewhere, and a colectomy was done. The colon was entirely studded with polyps.

(Slide) This patient is a man 73 years of age who presented himself with an advanced carcinoma of the rectum, which was removed by abdominoperineal resection. We gave a barium enema, and this patient taught us to give not only a barium enema but also a contrast enema.

On the portion of the bowel protruding through the left side we removed a polyp that was in this protruding portion of the descending colon. The polyp was malignant. We made further investigation of the colon, and subsequently it was removed, and this shows the specimen. (Slide)

(Slide) This slide is that of a woman 39 years of age who in January of this year underwent colectomy for a malignant lesion. She came to our community because her mother was in the hospital convalescing from a colectomy for a malignant lesion of the sigmoid. She spoke to us and told us of her previous operation and said that recently she had been passing some blood.

We examined her and found a lesion. We performed a biopsy, as we were able to approach it through the sigmoidoscope. We performed a colectomy low on the left side with primary anastomosis about six weeks ago. This case demonstrates that if there are associated polyps in the region of the malignant growth the extent of the operation can be expanded and those polyps can be removed along with the primary carcinoma. In order to determine this, one has to investigate the colon from the standpoint of contrast enemas and instrumentation from below. I believe that had the patient been examined by this method the scope of the operation could have been extended and the lesions could have been removed in one operation.

Incidentally, we had previously operated twice on a cousin of her mother for maligant lesion of the colon, colectomy having been performed on the right side in 1944 and on the left side in 1947. Undoubtedly, there were polyps present at the time of the first colectomy. We did not detect them by barium enema, and the patient subsequently returned. This illustrates the familial characteristics of polyps of the colon.

I have enjoyed this presentation because it is so practical.

DR. R. R. BEST, Omaha: I do not believe that I agree with all that has been said about sacrificing the sphincter mechanism in patients with polyposis of the colon. We are all acquainted with the procedure of anastomosing the ileum to the sigmoid in cases of polyposis of the colon after removal or destruction

of the polyps in the rectal and lower sigmoid regions with the electric current. It is another problem when polyposis of the colon plus carcinoma of the rectum is present, and in some of these cases one must sacrifice the sphincter mechanism. In some cases the polyps may be destroyed and then colectomy, in which the carcinoma is removed, may be accomplished, followed by anastomosis of the ileum to the rectal stump. About a year ago I reported on the experiences in 9 cases in which the ileum had been anastomosed to the anus or the lower rectal segment, and I must add that there is considerable difference between anastomosis of the ileum to the anus or the lower part of the rectum and anastomosis of the ileum to the sigmoid. This operation was originally planned for carcinoma of the rectum. At that time I was routinely removing the upper part of the sigmoid and even part of the descending colon in abdominal-perineal resection. Interested in preserving the sphincter mechanism and aware that downward spread in rectal carcinoma was unusual at a distance more than 2 cm. below the lower margin of the malignancy, and being unable to approximate the short rectal stump to the short descending colon, I transected the terminal end of the ileum and anastomosed it to the rectum or anus. The remaining part of the colon opened onto the abdominal wall as a small mucous fistula. The experiences which I had were not satisfactory, and my report was rather pessimistic. I did conclude that the operation of ileorectostomy or ileoproctostomy should be seriously considered for patients with polyposis of the colon and a malignant growth of the rectum. A permanent ileostomy may be avoided in some of these patients by this procedure. I do not believe that the operation of ileorectostomy or ileoproctostomy should be done on patients with carcinoma of the rectum unassociated with polyposis or in patients with ulcerative colitis. Therefore, in cases of polyposis of the colon associated with carcinoma of the rectum this procedure may be used and a permanent ileostomy avoided.

Dr. Roger Vaugan, Chicago: Some five or six years ago I started one of our residents on the problem of graphing the intestinal sounds in 1,000 normal persons. About the twelfth case I discovered during a conversation that the patient suffered from multiple polyposis of the large bowel; he had been everywhere, and the only opinion he could get as to what to do with it was to have it resected.

Being a young man and because there was no evidence of any malignant process as yet, he was reluctant to have this resection done. He had had the polyps in his rectum fulgurated as far as the sigmoidoscope would reach, and he inquired of me whether I could think of any method by which he might avoid a colonic resection so long as no growths of carcinomatous aspect had yet appeared.

I happened to remember the old Rovsing gastroscope and how it was inserted in the stomach to hunt for the bleeding point of an ulcer through a suture-encircled opening, and I said: "I wonder whether you could introduce an esophagoscope or a sigmoidoscope through such a puncture procedure, and fulgurate these young lesions. You would need an incision through the sigmoid, and you would need an incision in the transverse colon. I do not know just how you would handle the flexures."

He was so intrigued that he forgot all about peristaltic sounds and went after polyps, and he used all our local endoscopic tools at the postgraduate school on dogs and cadavers and had a lot of roentgenograms taken of the tubes in situs. The upshot of it was that he wrote a paper which was accepted by the Chicago Surgical Society as the best original contribution for the year, and he received the annual $500 prize of the society.

I have never had a case in which I could use this technic, and whether he ever had it used on himself or not I do not know. Certainly I did not do it. I offer this suggestion here since some of you will have such cases, and if you are intrigued with the idea, no doubt Dr. Cole can furnish the reference, since he has access to the Chicago Surgical Society's records.

# SITUS INVERSUS TOTALIS

## A Statistical Review of Data on Seventy-Six Cases with Special Reference To Disease of the Biliary Tract

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"SITUS inversus viscerum" is a term used to designate an anomalous condition in which organs or systems are transposed from their normal sites to locations on the opposite side of the body. It may include complete transposition of both the thoracic and the abdominal viscera or of only one or the other.

Fabricius [1] in 1600 reported the first known case of reversal of the liver and spleen in man, and Kuchenmeister [2] in 1824 was the first to recognize the condition in a living person. To Vehsemeyer,[3] in 1897, is given the credit for being first to demonstrate, by roentgen ray, transposition of the viscera.

During the years 1910 through 1947, of 1,551,047 patients registered at the Mayo Clinic, 76 were found on physical or roentgenologic examination, or both, to have a situs inversus of both thoracic and abdominal viscera. Seven others showed thoracic transposition only, and 4 more had transposition of the viscera that was confined to the abdomen. This gives an incidence of 0.00489 per cent for complete situs inversus, or 1 in 20,408 registrations. In the same period the condition was noted three times in 19,287 necropsies.

Adams and Churchill [4] in 1937 found an incidence of complete situs inversus of 0.002 per cent in the patients admitted to Massachusetts General Hospital and an incidence of 1 in more than 8,000 necropsies performed there.

In 1923 Guenther [5] gave the incidence of situs inversus as 0.0079 per cent, determined on the basis of clinical evidence, and as 3 in

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From the Division of Surgery, Mayo Clinic.

Read at the Fifty-Sixth Annual Meeting of the Western Surgical Association, St. Louis, Dec. 3, 1948.

1. Fabricius, cited by Wood and Blalock.[10]
2. Kuchenmeister, cited by Wood and Blalock.[10]
3. Vehsemeyer, cited by Wood and Blalock.[10]
4. Adams, R., and Churchill, E. D.: Situs Inversus, Sinusitis, Bronchiectasis: A Report of Five Cases, Including Frequency Statistics, J. Thoracic Surg. **7**:206-216 (Dec.) 1937.
5. Guenther, H., cited by Adams and Churchill.[4]

22,000 routine necropsies. Cleveland [6] in 1926 noted 1 example of complete situs inversus in 10,000 dissections, and Rösler [7] in 1929 recorded 3 instances of complete situs inversus in 22,402 necropsies.

Cockayne [8] gave the ratio of males to females with complete transposition as 32:27. In our group there were 40 males and 36 females. The youngest was 6 months old and the oldest 76 years.

For only 2 of the 76 patients was there any family history of situs inversus. Two men stated that they each had a brother (not a twin) with the same condition. Cockayne maintained that transposition of the viscera is caused by a single mendelian autosomal recessive characteristic. However, Torgersen [9] found only 6 instances of the condition in 573 siblings of patients with complete situs inversus. If a mendelian recessive characteristic had been present, the expected incidence would have been 1 in 3 and not 6 in 573. According to Torgersen, there have been 6 instances in which transposition occurred in 3 siblings and 19 instances in which the condition occurred in 2 siblings. Only 1 instance has been reported in which both mother and daughter were so affected.

Cleveland lists three theories as to the cause of the condition: 1. It results from the fact that persistence of a right omphalomesenteric vein has influenced the shifting of the stomach to the right instead of to the left. 2. It is due to the influence of the umbilical cord. In situs inversus, the cord is wound spirally to the right instead of to the left. According to this theory, the fact that the blood column flows in a different direction is responsible for the turning of the heart to the opposite side. 3. The condition is the result of the turning of the embryo to the right side of the umbilical vessels instead of to the left, as it normally lies.

A more complete review of the theories as to the cause of situs inversus is given by Wood and Blalock,[10] Adams and Churchill [4] and

6. Cleveland, M.: Situs Inversus Viscerum: An Anatomic Study, Arch. Surg. **13**:343-368 (Sept.) 1926.

7. Rösler, H.: Beiträge zur Lehre von den angeborenen Herzfehlern: VI. Ueber die angeborene isolierte Rechtslage des Herzens, Wien. Arch. f. inn. Med. **19**:505-610, 1929-1930.

8. Cockayne, E. A.: The Genetics of Transposition of the Viscera, Quart. J. Med. **7**:479-493 (July) 1938.

9. Torgersen, J.: Familial Transposition of Viscera, with Preliminary Remarks on Genetics and the Correlation with Diseases of the Respiratory Tract, Acta med. Scandinav. **126**:319-322, 1946.

10. Wood, G. O., and Blalock, A.: Situs Inversus Totalis and Diseases of the Biliary Tract: Survey of the Literature and Report of a Case, Arch. Surg. **40**:885-896 (May) 1940.

Jordan and Kindred.[11] It is believed that there are two types of persons with transposition of the viscera. The first type is that of a completely normal mutant and the second that of a true monster in whom other stigmas of maldevelopment may be anticipated.

Since Siewert[12] in 1904 reported the first case of what has since been called "Kartagener's syndrome," it has been known that the incidence of bronchiectasis and sinusitis in patients with complete situs inversus is greater than that noted in the general population. In 1933 Kartagener reported on 4 patients who had the triad of situs inversus, sinusitis and bronchiectasis. In 1935 he[13] reported 6 additional cases of the syndrome to which his name has been given.

In 1937 Adams and Churchill, in a study of 23 patients with situs inversus, found that 5, or 21.7 per cent, of the patients had bronchiectasis. The incidence of bronchiectasis in the hospital population is 0.306 per cent.

Fourteen patients at the clinic had all three components of the Kartagener syndrome, namely, situs inversus, bronchiectasis and sinusitis. Three other patients had situs inversus with bronchiectasis alone, making a total of 17 patients who had bronchiectasis of 80 with situs inversus (76 situs inversus totalis and 4 situs inversus thoracis). Four of the 17 had apparently only a thoracic situs inversus. This would give an incidence of bronchiectasis of 21.25 per cent in the patients with situs inversus, a definite increase over the incidence in persons without situs inversus.

Two of the 76 patients with complete situs inversus were found to have active duodenal ulcers on roentgenologic examination. Both patients were treated medically.

Although these patients are few and far between in the general surgeon's experience, it is essential that their existence be kept in mind. Thirty-six of the 76 patients who had a diagnosis of complete situs inversus underwent surgical treatment at the clinic or elsewhere, an incidence of 47 per cent. At least 2 had had incisions made in the right lower quadrant for removal of diseased appendixes, only to have a second incision made on the left side because of the undiagnosed situs inversus. Sixteen of the 76 patients with complete situs inversus underwent appendectomy, of whom 4 were operated on at the clinic and 12 elsewhere.

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11. Jordan, H. E., and Kindred, J. E.: A Textbook of Embryology, New York, D. Appleton & Company, 1926, pp. 239-241.

12. Siewert, A. K., cited by Adams and Churchill.[4]

13. Kartagener, M.: Zur Pathogenese der Bronchiektasien; Bronchiektasien bei Situs viscerum inversus, Beitr. z. Klin. d. Tuberk. **83**:489-501, 1933; Familiäres Vorkommen von Bronkiektasien, ibid. **84**:73-85, 1933; Situs viscerum inversus und Polyposis nasi in einem Falle familiäres Bronchiektasien, ibid. **87**:331-333, 1935.

Seven of the 76 patients had biliary disease. Two of the 7 were infants who had congenital atresia or obliteration of the bile ducts associated with complete situs inversus. Smyth[14] in 1941 reported the only other case we were able to find of congenital obliteration of the bile ducts associated with total visceral transposition.

The first infant was a boy aged 11 months who was admitted on Apirl 26, 1937, because of persistent jaundice. His delivery had been uncomplicated and at term. The birth weight was 7 pounds and 4 ounces (3.3 Kg.). His parents and 4 siblings were living and well. No situs inversus or other congenital anomaly was noted in them. The jaundice was present at the time of the child's birth, and it became progressively severe during the succeeding eleven months. The stools had been white since birth. The baby nursed poorly and was drowsy and listless all the time. Physical examination at birth showed micrognathia, dextroposition of the heart, transposition of the abdominal viscera and jaundice.

At the age of 2 months the child had had a short episode of fever and vomiting, but a diagnosis had not been made. At that time, marked enlargement of the liver was noted.

Physical examination at the time of his admission at the clinic showed a scrawny icteric male infant who weighed 11½ pounds (5.2 Kg.), as compared with the normal of 20 pounds and 8 ounces (9.3 Kg.). Dextrocardia was noted. The liver was firm and hard, and it extended 2 cm. below the left cos.al margin. The spleen extended 1 to 2 cm. below the right costal margin.

On May 5, 1937, choledochoduodenostomy was performed at the clinic. A primary incision in the upper part of the left rectus muscle was used. At operation a complete situs inversus of the abdominal viscera was found. The liver was nodular, greenish and about twice the normal size. The gallbladder was rather small and collapsed, and it did not seem to contain any bile. There was congenital atresia of the lower end of the common bile duct. The child died suddenly about twenty-eight hours postoperatively. The necropsy showed cirrhosis of the liver, with congenital atresia of the bile ducts in an infant with complete situs inversus.

The second infant was a girl aged 6 months who was admitted on July 14, 1947, because of progressive jaundice which had been present since the age of 9 days. Her delivery had been uncomplicated and was at term. Her parents, her 6 siblings and other members of the family did not have situs inversus or any other congenital anomaly.

On July 22, 1947, an operation was performed at the clinic. Through a primary incision in the upper part of the left rectus muscle the liver was found to be completely on the left side of the abdomen. It was grossly cirrhotic, and microscopic examination of hepatic tissue showed chronic hepatitis and fibrosis. No evidence of a gallbladder or of any extrahepatic or common bile duct system was found. A diagnosis was made of congenital atresia of the intrahepatic and extrahepatic biliary duct system. The incision was closed after further exploration of the abdomen revealed the situs inversus to be complete. The child had an uneventful postoperative course and was dismissed on Aug. 2, 1947, the eleventh postoperative day. We have not heard from her parents since her dismissal.

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14. Smyth, M. J.: Congenital Obliteration of Bile Ducts with Total Transposition of Viscera, Brit. M. J. **1**:84-85 (Jan. 18) 1941.

In 1940 Wood and Blalock found records in the literature of 17 cases of situs inversus totalis associated with disease of the biliary tract. They reported 1 of their own. In 1942 Metheny, Sherwood and Zimmerman [15] added a case.

Five of the 76 patients reported on here had had a diagnosis of cholecystitis and cholelithiasis. Two of the 5 had undergone cholecystectomy elsewhere prior to their registration at the clinic.

The first patient was a housewife aged 35 years who came to the clinic in 1918 because of a small dermoid cyst on the right brow. Her cholecystectomy was reported on by Hupp [16] in 1911. She stated that she had had no further abdominal complaints after her operation, at which complete transposition of the abdominal viscera had been found.

The second patient was a housewife aged 48 years who had had a cholecystostomy performed elsewhere in 1927. At that time a complete transposition of the abdominal viscera was found. She came to the clinic because of jaundice but refused further surgical treatment. She has not answered letters of inquiry as to her progress. To the best of our knowledge, this case has not been reported.

Three patients had a diagnosis of cholecystitis and cholelithiasis made at the clinic.

A retired farmer of Swedish descent, aged 59 years, came to the clinic on March 16, 1942, for a general examination. In 1934 an incision had been made on the right side of his abdomen because of the diagnosis of appendicitis, made elsewhere. No appendix was found. In 1936, as a result of a continuation of the attacks of appendicitis, an incision was made, also elsewhere, on the left side of the abdomen and he had undergone appendectomy. At that time, situs inversus of the abdominal viscera was found. He stated that his health had been good since his operation but that for the past two years he had noted that fatty foods caused "gas." His family history did not reveal situs inversus or any other congenital anomaly. Physical examination showed an essentially normal condition except for the scars of the operation mentioned previously and bilateral inguinal hernias Roentgen examination showed dextrocardia and situs inversus of the colon. The roentgenogram of the abdomen showed, in the left upper quadrant, a shadow which was believed to be a stone in the gallbladder. Surgical treatment was not advised, and the patient was dismissed on March 7, 1942.

The second patient with complete situs inversus for whom the diagnosis of cholecystitis and cholelithiasis was made at the clinic was reported on by Counseller [17] in 1931. We wish to report on the third

15. Metheny, D.; Sherwood, K. K., and Zimmerman, B.: Complete Situs Inversus Associated with Cholelithiasis, West. J. Surg. **50**:254-257 (May) 1942.

16. Hupp, F. L.: Malpositions of the Liver, New York State J. Med. **94**:422-426 (Aug. 26) 1911.

17. Counseller, V. S.: Rupture of the Spleen: Gunshot Wounds of the Abdomen; Transposition of Organs; Report of Cases, Proc. Staff Meet., Mayo Clin. **6**:501-505 (Aug. 26) 1931.

patient with that diagnosis at the clinic, who was the second to undergo cholecystectomy.

The patient was a Jewish housewife aged 46 years who had been born in Russia. As far as she knew, no other members of her family had had situs inversus or any other congenital anomaly. In 1934 appendectomy and suspension of the uterus had been done elsewhere. At that time transposition of all the abdominal viscera was found. Except for this, her history had been uneventful until about six months before registration at the clinic on May 24, 1948. Then she noted dyspepsia caused by ingestion of fatty food and the onset of attacks of epigastric pain which extended through to the back. These attacks occurred two to three times a week and at one time required a hypodermic injection of morphine for their relief. She never had been jaundiced. The previous diagnosis of complete situs inversus was confirmed by physical examination, which otherwise revealed nothing remarkable. The roentgenologic examination showed dextrocardia and a functioning gallbladder that contained multiple stones. The gallbladder was in the left upper quadrant of the abdomen. The laboratory examination of the blood and urine gave essentially negative results. Because of the history and findings, surgical treatment was advised. On May 29, 1948, one of us (C. W. M.) performed cholecystectomy and oophorectomy on the right side, utilizing an incision retracting the upper part of the left rectus muscle. All the organs in the abdominal cavity were found to be reversed. The stomach was on the right side and the gallbladder and liver on the left side. The liver was sharp edged. The stomach and pylorus were normal. The common bile duct was exposed down to the cystic duct, and the gallbladder was removed after a double tie had been placed on the cystic duct. The gallbladder fossa was closed. Exploration of the pelvis revealed a dermoid cyst of the right ovary, which was removed. The patient had an uneventful postoperative course and was dismissed on June 10, 1948, the thirteenth postoperative day. At that time she stated that she felt well.

## SUMMARY AND CONCLUSIONS

Seventy-six of 1,551,047 patients who registered at the Mayo Clinic from 1910 through 1947 were found to have complete (abdominal and thoracic) situs inversus on the basis of physical or roentgenologic observations or both. This represents an incidence of about 1 in 20,000. Forty-seven per cent of these 76 patients underwent surgical treatment at the clinic or elsewhere.

Two cases of congenital atresia of the bile ducts in infants with complete situs inversus are reported.

Five patients with complete situs inversus had had disease of the gallbladder previously or at the time of admission to the clinic. Two of the 5 had undergone cholecystectomy elsewhere; 1 of these was reported on elsewhere. One of the 5 did not have sufficient difficulty to warrant an operation. The other 2 of the 5 patients underwent cholecystectomy at the clinic, 1 of whom has been reported on previously; the other was the basis for a case report that is included here.

The possibility that situs inversus may be present must always be kept in mind by the physician performing abdominal surgery.

## DISCUSSION

DR. W. J. POTTS, Chicago: It is well to remember that congenital anomalies are frequently multiple. When an infant or newborn child presents a congenital anomaly which is obviously remediable surgically, one should look for other anomalies. It is embarrassing to correct an anomaly and find that one more serious has been overlooked.

Complete situs inversus is a congenital anomaly, whether it is total or whether it is just of the abdomen. I think that the reason that Dr. Mayo reports only 76 cases from their huge number of admissions is that they see relatively few newborn children. Many of the youngsters with transposition of the organs have other anomalies incompatible with life. During the past year we have seen 4 patients with situs inversus totalis. The first was a child who also had multiple congenital anomalies of the heart, including transposition of the great vessels.

(Slide) This otherwise normal child had just one anomaly—it was a mirror image of itself. It is a 3 month old baby who died about two weeks ago of meningitis. You see the heart on the right side and the gallbladder down on the left. It is a complete situs inversus, and this is the only child who did not have associated anomalies.

(Slide) This 4 month old youngster had complete transposition and exstrophy of the bladder. Besides, there was a prolapse of the rectum present since shortly after birth. Incidentally, we have seen 2 others like this in previous years. In each we have drawn down the prolapsed bowel until it was on slight tension, amputated the protruding portion and done an end to end anastomosis just proximal to the mucocutaneous line. It has been successful in 3 children; in each a competent sphincter has developed. Later the ureters were transplanted in this child.

(Slide) This child is rather unique—an 8 year old girl who was extremely cyanotic and had a tetralogy of Fallot. The heart was on the right side, typically boot shaped. Oddly enough, this child had still another anomaly in that the arch of the aorta was on the left. Of patients with tetralogy of Fallot on whom we have operated, 28 per cent, or 38, have had right aortic arches when the heart was on the left. This child had a mirror image of that—the heart on the right side and the arch of the aorta on the left. A typically aortic-pulmonary anastomosis was successfully done.

Congenital anomalies are multiple. Do not forget it, or you will suffer the same embarrassment which I have experienced.

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|---|---|---|---|
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| **INSTEAD OF** | **INSTEAD OF** | **INSTEAD OF** | **INSTEAD OF** |
| Mayo Intestinal #1, #2<br>Murphy Intestinal #1, #2<br>Ferguson #6, #8<br>Mayo Catgut #1, #2 | Mayo Intestinal #3, #4<br>Murphy Intestinal #3, #4<br>Ferguson #10, #12<br>Mayo Catgut #3, #4 | Regular Surgeon's #2, #3<br>Fistula #2, #3<br>Mayo Trocar #2<br>Martin's Uterine #4 | Regular Surgeon's #4, #5<br>Fistula #4, #5<br>Mayo Trocar #3, #4<br>Martin's Uterine #5, #6 |
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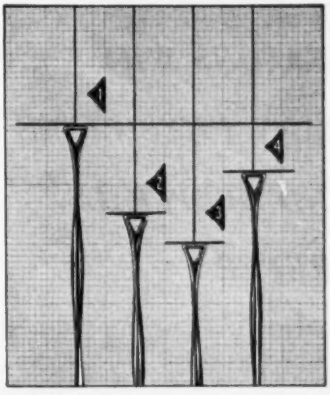


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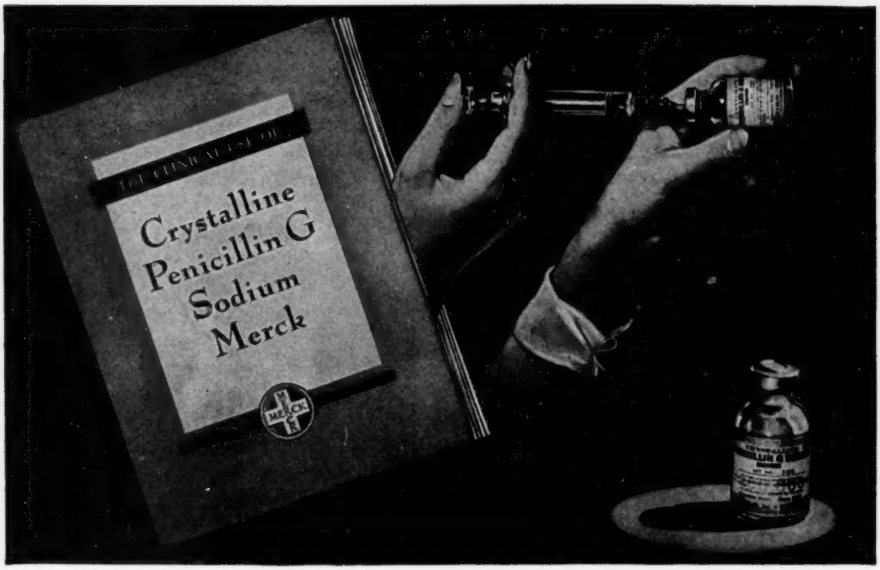


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